“A STUDY ON CLINICAL PROFILE OF RIGHT VENTRICULAR MYOCARDIAL INFARCTION”

DISSERTATION SUBMITTED FOR

M.D. DEGREE EXAMINATION

BRANCH I GENERAL MEDICINE

of

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY

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I hereby certify that this work embodied in the dissertation **"A STUDY ON CLINICAL PROFILE OF RIGHT VENTRICULAR MYOCARDIAL INFARCTION"** is a record of work done by **Dr.RAJAGOPAL.S.** in the Department of general medicine, Tirunelveli Medical College, Tirunelveli, during his postgraduate degree course in the academic period 2010-2013. This work has not formed the basis for any previous award of any degree.

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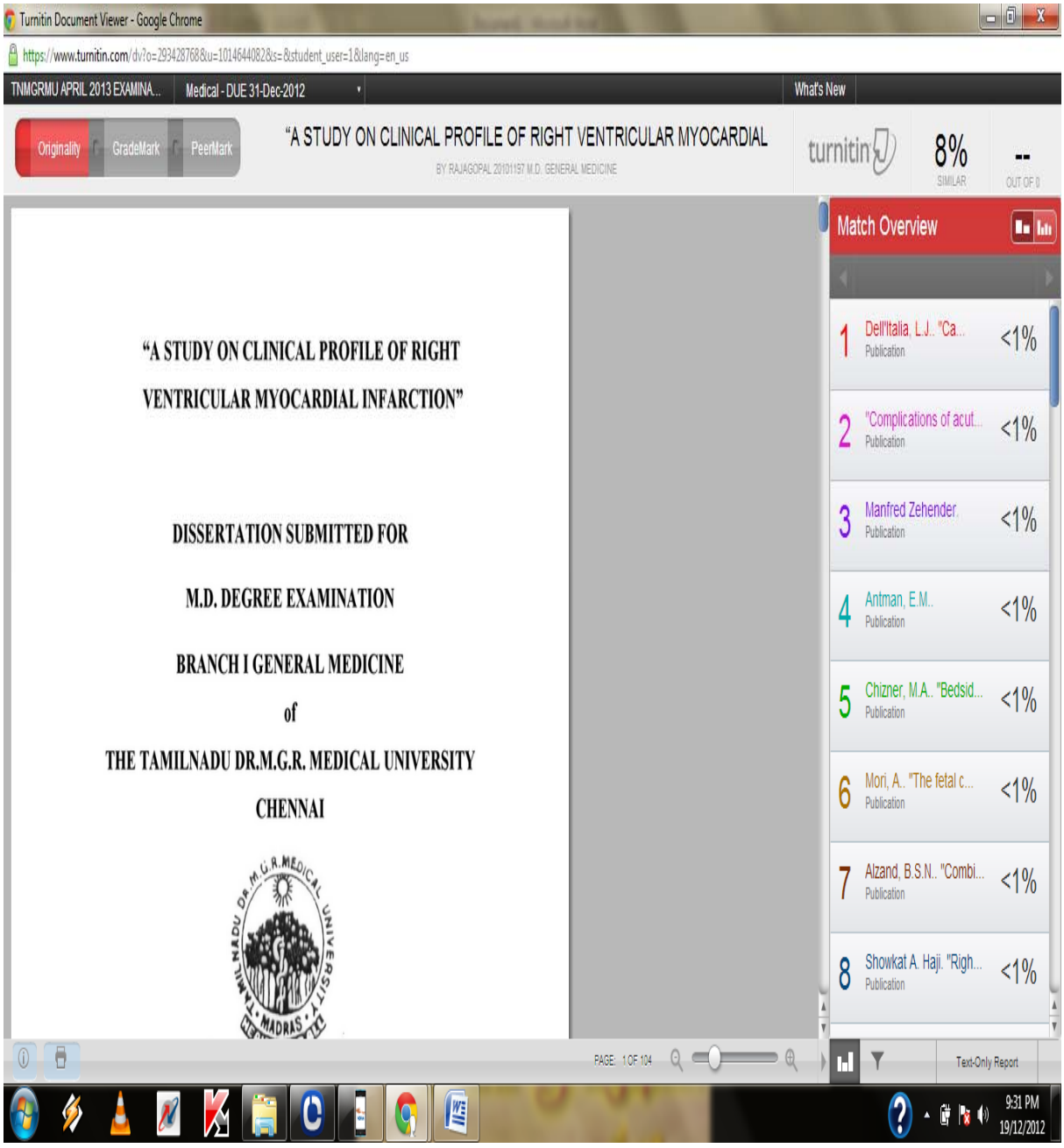
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DECLARATION

I solemnly declare that the dissertation titled **"A STUDY ON CLINICAL PROFILE OF RIGHT VENTRICULAR MYOCARDIAL INFARCTION"** is done by me at Tirunelveli Medical College Hospital, Tirunelveli.

The dissertation is submitted to The Tamilnadu Dr. M.G.R. Medical University towards the partial fulfilment of requirements for the award of M.D. Degree (Branch I) in General medicine.

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CONTENTS

INTRODUCTION

Cardiovascular diseases (CVD) comprise of diseases of heart and vascular system. Worldwide CVD is the most common cause of death due to non-communicable disease, which is about 17 million. More than one third of these deaths occur in middle aged adults. In developing countries, it is responsible for about one third of such deaths [1]

CAD is a spectrum of diseases consisting of stable angina pectoris and acute coronary syndrome, which includes Unstable angina, non ST elevation MI(NSTEMI) and ST elevation MI (STEMI). Data showed that 25-28 % of patients who had acute myocardial infarction die suddenly. Of about 55 % of all cardiac deaths occur within the first hour.

Of STEMI, the incidence of isolated right ventricular myocardial infarction is very rare which is found to be < 3%. It is usually associated with IWMI. Inferior wall myocardial infarction has high mortality when it is associated with RVMI while comparing to the mortality of isolated inferior wall myocardial infarction (25-30% vs. 6%). So, early recognition of right ventricular myocardial infarction is important to reduce the increased complications and death in IWMI, when it is seen with RVMI. RVMI has high mortality in elderly individual. [2]

Moreover, the management of RVMI is also entirely different from other types of myocardial infarction. In RVMI, the treatment consists of

achieving adequate preload by intravenous infusion of normal saline, maintaining normal haemodynamic parameters by intravenous infusion of inotropic agents and maintaining of normal rhythm by drugs or cardiac pacemakers. All these measures should be undertaken in addition to the emergency life saving measures and reperfusion therapy, whenever indicated. In contrast to RVMI, reperfusion therapy is the main treatment in other types of myocardial infarction, in which volume loading may lead onto worsening of the cardiac status.

Also, the acute outcome and the long term prognosis in right ventricular myocardial infarction are higher than the other types of myocardial infarction. In right ventricular myocardial infarction, complete recovery of the cardiac status to normal or near normal level results within weeks to months following timely institution of adequate and appropriate measures.

AIMS AND OBJECTIVES OF THE STUDY

1. To study the incidence of isolated right ventricular myocardial infarction and

   in association with other types of myocardial infarction in Tirunelveli

   Government medical college hospital over a period of one year.
2. To study the typical and atypical symptoms of right ventricular myocardial

   infarction.
3. To study the various clinical features, risk factors, electrocardiographic

   features and echocardiographic features of right ventricular myocardial infarction.
4. To study the various complications and its correlation with clinical features

   of right ventricular myocardial infarction.
5. To study the occupational and socio economic status of patients presenting

   with right ventricular myocardial infarction.

REVIEW OF LITERATURE

CORONARY BLOOD FLOW

Blood supply to the heart occurs via two arteries. These are the left main coronary artery and the right coronary artery. Both arteries arise from the corresponding cusp in the root of aorta. Proximal part of right coronary artery supplies sinu atrial (SA) node and right atrium. Middle part of right coronary artery supplies infero- posterior, lateral part of right ventricle. Distal part of right coronary artery supplies atrio-ventricular node (AV node).Posterior descending branch supplies infero-posterior, lateral left ventricle and lower part of inter ventricular septum. In normal situations, the myocardium will be able to control the supply of oxygen rich blood in response to the increased demand and prevents the subsequent development of ischemia and infarction by various mechanisms.[3]

The normal coronary blood flow mainly depends on cardiac requirement of oxygen. The oxygen need of myocardium mainly depends on cardiac contraction, cardiac rate and stress of the myocardial wall. The unique chrecteristic of blood flow through the coronary arteries is that most of the blood flow occurs during diastole.

Most of the resistance (approximately 75%) to the coronary blood flow occurs at three levels. The first one is at the level of epicardial arteries (Resistance 1). The second one is at the level of the prearteriolar

vessels (Resistance 2). The third one is at the level of arterioles and intra myocardial capillaries (Resistance

3). Among the three levels, second (prearteriolar) and third level constitute most of the coronary resistance. The resistance occurring at the level of epicardial coronary arteries is trivial, if there is no significant obstruction due to atherosclerosis or some other reasons.

REGULATION OF CORONARY BLOOD FLOW

METABOLIC REGULATION

The coronary blood flow is controlled by changes occurring in the coronary vascular resistance according to the metabolic need of heart. The metabolic need of the heart depends on the level of exertion and the emotional stress of the individual.

AUTOREGULATION

This is the adaptation of the resistance of the coronary blood vessels according to the physiological changes in the blood pressure level, so as to give adequate blood flow to meet the cardiac oxygen requirement.

NORMAL RIGHT VENTRICLE

Venous blood reaches the right ventricle at low diastolic pressure and with more respiratory fluctuation when compared to the left ventricle. Normally right ventricle has $1/3^{rd}$ of thickness of wall and $1/6^{th}$ of muscle mass when compared to the left ventricle. Also it pumps out the venous

blood into the low resistant pulmonary vascular system. Pulmonary vascular system has 1/5th of systemic arterial pressure, so it offers less resistance (1/10th of systemic arterial) to right ventricular cardiac output[4] .The pattern of contraction of left ventricle muscle occurs in radial and longitudinal direction. But in right ventricle the pattern of contraction is different. Free wall of right ventricle contracts towards inter ventricular septum. The interventricular septum contracts in longitudinal direction and forces the blood towards the right ventricle. So, some amount of right ventricle function is contributed by inter ventricular system. This contribution of interventricular septal contraction to the normal right ventricular function becomes significant in the setting of right ventricular myocardial infarction involving free wall of right ventricle. The normal RV functions smoothly even in the presence of changes in the preload. But in the presence of ischemia, right ventricle becomes highly sensitive to changes in the preload. For example, in right ventricular infarction, right ventricular failure occurs when the patient is given morphine, diuretics, nitrates or beta blockers. All these drugs reduces right ventricular preload.

RIGHT VENTRICLE WITH INFARCTION

Significant hemodynamic disturbances occur when more areas of right ventricle become infarcted. So, significant hypotension occurs with more proximal occlusion of right coronary artery before it gives off acute

marginal branches; Because, such a type of occlusion leads to infarction of right ventricular free wall and posterior wall. The compliance of right ventricle is profoundly affected by right ventricular infarction. So it becomes more dependend on preload for adequate right ventricular filling. In this situation right atrial contraction significantly contributes to right ventricular filling by pushing blood into the less complaint right ventricle[5]. This contribution was lost with right atrial infarction and atrial fibrillation. Atrial fibrillation occurs with more proximal occlusion of right coronary artery before sinus nodal branch arises. If the infarction also involves the interventricular septum, contribution of interventricular septum to the right ventricular function is lost.

As right ventricular output becomes reduced, less blood goes to the left ventricle. So left ventricular filling is reduced. Since the right ventricle is a thin walled structure, it becomes dilated in right ventricular infarction and leads to right atrial dilatation. This dilated right ventricle pushes the inter ventricular septum towards left ventricle. So left ventricular cavity size becomes reduced and leads to left ventricular dysfunction.

As heart is confined within closed pericardial structure, dilated right ventricle and right atrium compresses left ventricle and occupies most of the pericardial space leaving behind less space for left ventricular dilatation.

Although volume loading is the main treatment in right ventricular myocardial infarction, it actually reduces the cavity size of left ventricle by dilating the less complaint, infracted right ventricle.

The infracted right ventricular myocardium has steeper pressure - volume relationship. So it becomes highly sensitive to the changes in the preload. The use of preload reducing drugs like nitrate, morphine, diuretics in RVMI leads to hypotension.

Because of the reasons cited above, low output hypotension syndrome occurs commonly in RVMI.

The incidence of RVMI is less than that of left ventricle. Not all right coronary arterial occlusions produce right ventricular myocardial infarction. Also right ventricular myocardial infarction has better, complete and fast recovery than that of left ventricle [6]. This may be due to the following reasons.

1. Blood flow to the RV occurs both during systole and diastole.
2. Less wall thickness of right ventricle. So better passive diffusion of blood from right ventricular cavity to the right ventricular wall occurs.
3. Presence of thebesian vessels.
4. More collateral blood flow to the right side ventricle.
5. Less work in the presence of more nourishment.

Right ventricular infarction differs from left ventricular infarction in the following ways.

1. Right ventricular infarction is less common than in left ventricular infarction due to reasons as cited above.
2. Pre infarction angina protects against the right ventricular infarction [7].
3. Pre load determines the adequacy of right ventricle filling in the setting of RVMI
4. Contraction of interventricular septum and right atrium has prominent role in the right ventricular function and right ventricular filling respectively in RVMI
5. Pericardium mediated inter ventricular dependence becomes an active phenomenon in RVMI, due to rapid dilatation of right ventricle in RVMI
6. Right ventricular failure occurs in RVMI and left ventricular failure occurs in the left ventricular infarction.
7. Right ventricle has better and quicker recovery rate than left ventricle in the setting of infarction.

CAUSES OF CORONARY ARTERY DISEASE.

Atherosclerosis of the coronary arteries accounts for the more than 80% of cases of coronary artery disease. Atherosclerosis is the most

common type of arteriosclerosis. Arteriosclerosis literally means hardening of the arteries [8].

Other less common causes of myocardial ischemia are

1. Prinzmetal angina –due to coronary artery spasm
2 Aortic stenosis – due to reduced blood flow and increased oxygen demand due to left ventricular hypertrophy
3 Thrombo-embolism involving coronary arteries
4 Carotid artery dissection
5 Narrowing of coronary artery origin in aortitis
6 Unusual origin of LAD artery from pulmonary artery (common in infants)
7 Abnormal constriction or failure to normally dilate in response to increased demand –known as micro vascular angina
8 Severe anemia or the presence of carboxy hemoglobin- due to reduced oxygen carrying capacity

EPIDEMIOLOGY

Cardiovascular disease (CVD) is the most common cause of death worldwide, accounting for 40% of death in the developed counties and 28% in the developing countries like India. The global rise in CVD is due to profound changes in the causes worldwide morbidity and mortality in recent past centuries and has been termed as epidemiological transition.

The main reasons for the epidemiological transition are high level of industrialization, urbanization and its resultant life style changes. All the parts of the world are affected by this epidemiological transition without difference among the various races, cultures and ethnic groups. Worldwide cardiovascular disease caused about 32% of death in the year of 2000 and this death rate is expected to rise as 2 million with a 30% increase over the previous decade. Most of the cardiovascular death occurred in India in the 35-64 age group. In India this death rate is expected to rise to 17.9 million by 2030.

In India coronary artery disease increased by nine fold in urban areas and two fold in rural areas. In India coronary artery disease has become the most common non communicable disease nowadays. Coronary artery disease occurs more commonly in the low socioeconomic people at a rate, which is equal to or more than that of socio economically developed people. India has reached a stage in global epidemiological health transition, which is characterized by more decrease in the infectious, nutritional diseases (pre-transitional disease) and profound increase in the non communicable disease particularly coronary artery disease (post-transitional disease) .World bank estimation shows that it is expected that cardiovascular death will become single most common cause of death in India by the year of 2015. It also pointed out that the rate of rise in

coronary artery disease will further increase by many fold in the near future if the risk factors persist at the same level and appropriate preventive measures are not taken.

The factors responsible for rapid rise of coronary artery disease in India include demography shift, change of life style pattern, change in the dietary pattern, reduced intake of fresh fruits and green leafy vegetables (which is cardio protective) , and the epidemiological health transition [9]. Many authorities stated that in Indians CAD occurs in severe form and prematurely. Also it was found that 52.2% of Indians are having death at below the age of 70 years, which is 22.8% in western countries. More data is available to support the fact that Indians are more vulnerable to be affected by CAD. SHARE study has proven this.

This is because of higher incidence of dyslipidemia, higher body fat content, higher prevalence of diabetes& insulin resistant syndrome and higher prevalence of novel risk factors like, lipoprotein (a), homocystein, fibrinogen level, plasminogen activator inhibitor-1 level. Higher level of infection, low birth weight and low level of antioxidant also contribute to the rise of CAD in India.

Coronary artery disease in women has some special features [10]. These include, latter age of presentation (Post menopausal age is the risk factor for coronary artery disease in women), stronger influence of

diabetes, atypical presentation of coronary artery disease, higher chances for death following first myocardial infarction, higher long term morbidity, less evidence for lipid lowering therapy and hormone replacement therapy in women.

In addition to the conventional and novel risk factors, right ventricular myocardial infarction has some special risk factors. These include right ventricular hypertrophy of any cause and iatrogenically induced RVMI. In India, RVH occurs commonly secondary to chronic obstructive pulmonary disease. Smoking is the most common risk factor for chronic obstructive pulmonary disease. It is also the major risk factor of acute myocardial infarction in Indian patients. The iatrogenic cause of right ventricular myocardial infarction includes any cardiac intervention which involves right coronary artery. In both of these situations reduced cardiac blood flow in the face of increased demand resulting in right ventricular myocardial infarction.

CLINICAL FEATURES.

PRECIPITATING FACTORS.

The common precipitating factors include heavy exercise, intense emotion, exposure to cold, heavy meals intake, undertaking of unfamiliar task and non-cardiac surgical procedures.

Reduced myocardial perfusion secondary to hypotension of any cause and increased myocardial oxygen demand caused by aortic stenosis, fever, tachycardia, respiratory infections, psychological stress, hypoxemia of any cause, pulmonary embolism, hypoglycemia, serum sickness, allergy, rarely wasp stings and drugs like ergot alkaloids, cocaine, sympathomimmetics are the remaining precipitating factors. But acute myocardial infarction can occur without any of these precipitating factors.

More than 50% of the patients with right ventricular myocardial infarction patients found to have a precipitating factor and prodromal symptoms.

CHEST DISCOMFORT

The typical symptom of acute myocardial infarction is sudden onset of persistent, precardial or retrosternal discomfort, which is commonly described by the patients as a pressure, crushing, aching, or burning sensation. Radiation to the neck, back, or arms common (left arm > right arm). The discomfort reaches maximum intensity over short period (crescendo pattern). Usually associated symptoms are nausea, diaphoresis, generalized weakness, anxiety and a fear of impending death. Of about 20 % of patients are presenting with atypical symptoms or asymptomatic. Atypical presentations include cardiac failure (dyspnoea without crescendo type of chest pain), classic angina pectoris without a severe or prolonged

period, atypical location of the pain, ¸central nervous system features like stroke, mania or psychosis, giddiness and syncope and acute indigestion[11].

Painless myocardial infarction is commonly seen in elderly, women, diabetic patients, and in postoperative patients. These patients may present with dyspnoea or congestive heart failure. In inferior wall myocardial infarction, patients commonly present with nausea and vomiting .This may be due to parasympathetic fiber stimulation or as a part of Bezold zarisch reflex. The Bezold zarisch reflex is due to the stimulation of vagal fibers, situated in inferior part of left ventricle. Right ventricular MI is more commonly associated with inferior wall MI. So, gastro intestinal symptoms like nausea, vomiting, indigestion, epigastric pain are more common with right ventricular MI also. Symptoms of hypotension like giddiness, syncope are more common in myocardial infarction involving inferior wall and right ventricle, than infarction involving anterior wall, due to the more frequent association of hypotension. But dyspnoea is less common in right ventricular myocardial infarction due to the absence of pulmonary congesion, which is the one of the characteristic clinical triad of right ventricular myocardial infarction.

CIRCADIAN VARIATION

Most cases of acute myocardial infarction have the onset of symptoms between 1.00 A.M. to 12.00.P.M. This type of circadian variation may be due to variation in the activity of autonomic nervous system. Sympathetic nervous system is more active in the early morning hours, so the resultant surge in the blood pressure and heart rate will be responsible for the higher incidence of acute myocardial infarction during early morning hours.

ATTITUDE AND APPEARANCE OF PATIENT

Patient with acute myocardial infarction will be restless, anxious, anguished facial expression .They will show their clenched fist against their chest wall to describe the chest discomfort. This is known as Levin's sign. Patients with left ventricular failure will be dyspnoeic at rest, in the propped-up position to get easy breath and have cough with pink colored sputum due to acute pulmonary edema. These patients also have pallor of the skin and face, cyanosis involving lips and nail beds, cold perspirations of skin due to hypoxia and peripheral vasoconstriction due to sympathetic stimulation. They will be in a drowsy state, confused, disoriented or in an unconscious state depending upon the level of cerebral hypo perfusion. These features of hypotension and shock are more common in RVMI, as

severe hypotension and shock are often the presenting features of this entity.

HEART RATE

The rhythm may be regular or irregular. It may be normal(at the rate of 60 to 100 per minute) or bradycardia(at the rate of < 60 per minute) or tachycardia (at the rate of > 100 per minute). Bradycardia may be due to sinus bradycardia, sinus node dysfunction, atrio-ventricular nodal block. Although bradyarrythmias occur in all type of myocardial infarction, they are more common in inferior wall and right ventricular myocardial infarction. This may be due to atrial infarction, ischemia to sinus node and atrio ventricular node, ventricular septal rupture due to septal infarction, or due to increased vagal stimulation as a part of Bezold- zarisch reflex.

Regular tachycardia may be sinus tachycardia, supra ventricular tachycardia or ventricular tachycardia. Irregular tachycardia may be ectopic beats, atrial fibrillation or ventricular fibrillation. Sinus tachycardia occurs due to chest discomfort, anxiety, and hypoxemia or due to drugs like morphine . It comes to normal rate once pain subsides. Sinus tachycardia is more common in anterior wall MI and in patients presenting with cardiac failure. Atrial fibrillation commonly occurs in right ventricular MI due to atrial infarction. Ectopic beats, ventricular tachycardia and ventricular fibrillation can occur in all type of myocardial infarction.

BLOOD PRESSURE

Patients with uncomplicated myocardial infarction usually have normal blood pressure. Hypotension and shock can occur in all patients presenting with cardiac failure, inferior wall and right ventricular myocardial infarction. In inferior wall and right ventricular myocardial infarction, hypotension will be aggravated by assuming trendelenburg position and preload reducing drugs like morphine, nitrates, and diuretics. In MI involving inferior wall, the reason for hypotension is excessive vagal stimulation[12]. In right ventricular myocardial infarction, hypotension is mainly due to low cardiac output syndrome, which is the characteristic feature of MI involving right ventricle. Worsening of hypotension on administration of preload reducing drugs in RVMI is due to its increased sensitivity to alteration in preload. It is also one of characteristic feature of RV myocardial infarction.

Hypertension may be the presenting feature of anterior wall myocardial infarction due to increased sympathetic stimulation. When it is due to anxiety, fear, nervousness, it comes down to normal level once patient becomes comfortable. Previously hypertensive patients may have normotension or hypotension depending upon the extend of infarction.

TEMPERATURE

Although most of the patients with AMI usually have normal body temperature on admission, mild elevation of temperature may occur during the first week of MI due to systemic inflammatory response. Hypothermia is present in all patients presenting with shock, due to sympathetically mediated vasoconstriction and increased sweating. Because of frequent association of hypotension and shock in patients of inferior wall MI and patients of RV myocardial infarction, these patients frequently have hypothermia than others.

RESPIRATION

Increased respiratory rate in acute myocardial infarction is due to cardiac failure, anxiety and apprehension. Patients with right ventricular myocardial infarction will have normal respiratory rate or increased rate due to shock and poor peripheral perfusion.

JUGULAR VENOUS PRESSURE

Although jugular venous pressure is elevated in all myocardial infarction patient with shock, normal jugular venous pressure or flat neck veins will be seen in patients with hypotension, hypo perfusion or hypovolemia. JVP is elevated in patients with RVMI due to right heart failure. It is one of the characteristic clinical triad of right ventricular myocardial infarction. The triad of low output, elevated JVP and lack of

pulmonary stasis has high specificity (96%) and low sensitivity (25%) in the diagnosis of RV myocardial infarction in the presence of inferior wall myocardial infarction. The severity of hemodynamic disturbances of RVMI is clinically assessed by elevated jugular venous pressure alone with 88% of specificity and 69% of sensitivity in the presence of right ventricular MI. But by combining the elevated jugular venous pressure with kussmaul's sign, haemodynamically significant right ventricular myocardial infarction can be fairly diagnosed with accuracy of 100% specificity and 88% sensitivity with the presence of inferior wall MI. The RV myocardial infarction also shows some specific features in jugular venous pulse wave form.

A wave and X descent[13]

Right ventricular myocardial infarction shows prominent A wave and X descent and diminished Y descent when patient is in normal sinus rhythm due to augmented right atrial contraction against failing dilated, failing right ventricle. If patient is having atrial fibrillation due to atrial infarction, A wave, X descent and Y descent will be diminished with high right atrial and systemic venous pressure due to the loss of right atrial contraction. Diminished A wave and significant hemodynamic disturbance in a patient with right ventricular myocardial infarction points towards

significant right atrial infarction. It also indicates bad prognostic sign even in the presence of good left ventricular function.

Tall C-V wave

Right ventricular myocardial infarction shows tall C-V wave in jugular venous pulse wave form when it is complicated by tricuspid regurgitation due to papillary muscle dysfunction or rupture.

Cannon A wave

Right ventricular myocardial infarction exhibits cannon A wave if it is associated with complete heart block due to atrio ventricular node ischemia or ventricular septal rupture. In right ventricular myocardial infarction patients present with features of shock, jugular venous pulse will be obscured. In such a situation, jugular venous pressure will be made to be seen clearly by volume loading.

Kussmaul's sign

Normally jugular venous pressure will fall by at least 3 mm Hg during inspiration. A rise in jugular venous pressure (or failure to decrease) with inspiration is known as the kussmaul's sign, it occurs with right ventricular myocardial infarction .It is due to inability of failing dilated right ventricle to accommodate increased venous return, which occurs during inspiration. Kussmaul's sign is one of the characteristic clinical triad of right ventricular myocardial infarction. In the presence of inferior wall

myocardial infarction, kussmaul's sign along with elevated jugular venous pressure points towards the diagnosis of right ventricular MI with high accuracy.

CAROTID PULSE

Palpation of carotid pulse identifies RVMI patient with shock by its small volume of pulse due to reduced left ventricular stroke volume. Sharp and brisk upstroke of carotid pulse indicates ventricular septal rupture, which may occur as a complication of right ventricular myocardial infarction. Pulsus alternans indicates severe left ventricular dysfunction.

Pulsus alternance

Pulsus alternans is seen in patients presenting with cardiogenic shock. When pulsus alternans is associated with T-wave alternans in electrocardiography, the risk of arrhythmia is increased

EXAMINATION OF CARDIOVASCULAR SYSTEM

Palpation

Reveals palpable P2 and parasternal heave in a case of isolated right ventricular myocardial infarction due to right ventricular hypertrophy, which is secondary to pulmonary hypertension of any cause. Systolic thrill and diffuse apical impulse of ventricular septal rupture and papillary muscle dysfunction or rupture can be palpated. It also identifies dyskinesia, as a paradoxical bulge in the latter part of systole. Palpable right sided S4 is

felt in the tricuspid region in a case of right ventricular myocardial Infarction.

Auscultation

The following auscultatory findings appreciated in right ventricular myocardial infarction include muffled heart sounds of acute myocardial infarction, muffled S1 of inferior wall MI due to prolonged PR interval, crescendo-decrescendo mid- systolic murmur of papillary muscle dysfunction, harsh blowing pan-systolic murmur of ventricular septal rupture, pericardial rub due to pericarditis, right sided S3&S4 which heard in the left lower sternal area and increases on inspiration.

EXAMINATION OF OTHER SYSTEM

Auscultation of lung fields

Although clear lung field is the one of characteristic clinical triad of right ventricular myocardial infarction, bibasilar crackles will be heard in extensive RVMI involving left ventricle.

Per-abdominal examination

Reveals features of right heart failure, which is the cardinal feature of RV myocardial infarction. These are tenderness in the right upper quadrant of abdomen, congestive hepatomegaly, hepato jugular reflex & minimal ascities.

ELECTROCARDIOGRAPHIC FEATURES [14,15]

Electrocardiographic manifestations of right ventricular myocardial infarction varies with the level of right coronary arterial occlusion.

When occlusion occurs proximally (proximal to acute marginal branch) ischemia to the sinus node and atrial free wall results. It will lead to sinus bradycardia & atrial infarction, atrial fibrillation respectively.

When middle part of right coronary artery (part containing acute marginal branch) occluded, infarction of lateral and infero-posterior free wall of right ventricle occurs. This will result in ST-segment elevation in corresponding leads.

When distal segment of right coronary artery (distal to acute marginal branch) occluded, ischemia to the atrio-ventricular node results. This will leads to atrio-ventricular nodal block.

When occlusion occurs in the posterior descending part of right coronary artery, This will leads to infarction of inferior free wall & posterior part of left ventricle and inferior part of septum. This will manifest in the electrocardiography as ST segment elevation in corresponding leads.

Right ventricular myocardial infarction is manifested in the electrocardiography as ST segment elevation in V1-V4. This type of electrocardiographic picture will confuse with those of antero-lateral

myocardial infarction. So, this type of electrocardiographic pattern is known as pseudo antero-septal myocardial infarction. This is particularly true for isolated right ventricular myocardial infarction. Because in isolated right ventricular myocardial infarction, there is no ST segment elevation in inferior leads (lead 2,3,aVf).

This pseudo antero-lateral myocardial infarction pattern is differentiated from electrocardiographic pattern of right ventricular myocardial infarction by the following ways.

1. Right ventricular myocardial infarction is associated with ST segment elevation in inferior leads (if it is associated with inferior wall myocardial infarction)

2. ST segment elevation in V4R seen in right ventricular myocardial infarction

3. ST segment elevation in chest leads V1-V4 in right ventricular myocardial infarction is seen as follows.ST segment elevation in V1>V2>V3>V4. But in true antero septal myocardial infarction the pattern will be V1<V2<V3<V4.

4. The mean frontal plane ST vector in Isolated right ventricular myocardial infarction is directed towards >90 degree to the right side and producing ST segment depression in lead 1. But in true antero-septal

myocardial infarction the vector is directed towards -30 degree to -90 degree to the left side and producing ST segment elevation in lead 1

Other electrocardiographic patterns of right ventricular myocardial infarction includes

1. Ratio of ST elevation inV2 and ST segment elevation in aVF < 0.5 mm
2. Qs Complex in V4R
3. Absence of reciprocal changes in right sided chest leads in inferior wall myocardial infarction
4. ST segment depression in anterior chest leads
5. Positive T wave with ST elevation inV4R indicates proximal right coronary artery occlusion
6. Positive T wave without ST elevation in V4R indicates distal right coronary artery occlusion.
7. ST segment elevation >0.1mV in lead II+ III/II >1 and in lead III+ III/II >1
8. Presence of epsilon waves

EPSILON WAVES IN RIGHT VENTRICULAR MYOCARDIAL INFARCTION [16]

Epsilon waves are low voltage positive deflection between the end of QRS complex and starting of T wave. Their presence indicates delayed

activation of viable myocardium, which is present in between the infarcted myocardium. They are also seen in right ventricular hypertrophy, infiltrative diseases of right ventricle, and arrythmogenic right ventricular dysplasia. Because of their poor sensitivity and poor specificity, they are not used widely in the diagnosis of right ventricular myocardial infarction.

ST segment elevation in V4R >1mm is having 70% sensitivity and 100% specificity with predictive accuracy of 80% in diagnosing right ventricular myocardial infarction in the presence of inferior wall infarction. The degree of ST segment elevation in V4R is also the strong predictor of major complications of right ventricular myocardial infarction and its in-hospital mortality. But this elevation lasts only for less than 12 hours. ST segment elevation also seen in acute pulmonary embolism, left ventricular hypertrophy, acute anterior septal infarction, acute pericarditis, and previous anterior infarction with aneurysm. So, all the patients with inferior wall myocardial infarction should have right sided chest leads immediately after presentation, without delay for early diagnosis of right ventricular myocardial infarction. Apart from diagnosing right ventricular myocardial infarction, the electrocardiogram also plays role in identifying atrial infarction, atrial fibrillation, sinus bradycardia, right bundle branch block, left anterior hemi block, complete heart block, ventricular tachycardia ventricular fibrillation, right ventricular aneurysm.

CARDIAC BIOMARKERS

Cardiac biomarkers are nothing but enzymes present in the cardiac muscle. These include myoglobin, creatine kinase and its isoform CK-MB ,troponin I and troponin T, alanine aminotransferase, lactate dehydrogenase. These are released into the bloodstream when the cardiac muscle fibers are damaged. So, the acute myocardial infarction can be made by measuring the timing of rising of its blood level, rate of release, duration for which it persists in the blood.

Tests for cardiac biomarkers should be done on admission. If first test is negative, and the clinical suspicion of acute myocardial infarction is high, repeat test should be done at 6-9 hours and 12-24hours of admission.

TRPONIN[17]

They are more specific cardiac biomarker than any one. They are participating in muscle contraction by regulating actin and myosin interaction. There are two types of troponin. Troponin I and troponin T. Their molecular weight is 23000 and 33000 kDa respectively. They start to appear in blood within 12 hours of cardiac injury, attains highest level at 12-24 hours after cardiac injury. They remain detectable for 1-2 weeks in troponin I and 1-3 weeks in troponin T. Level of troponin in blood correlates with extent of infarction and correlate with prognosis.

COMBLETE BLOOD COUNT

The lower haemoglobin level and higher haemoglobin level adversely affect clinical outcomes in myocardial infarction patients due to anemia and polycythemia respectively. The total white blood cell count starts to rise within hours, attains its peak level within 4 days and comes to normal value within a week period. The more the count value, the more severe coronary artery occlusions will be present and associated with worst outcome. The haematocrit value starts to increase within hours to days due to haemoconcentration. The erythrocyte sedimentation rate raises within week and remains elevated for several weeks. It does not appear to correlate with morbidity and mortality. The elevated value of C-reactive protein appears to be associated with worse angiographic pattern in acute coronary syndromes.

LIPID PROFILE

Test for lipid profile should be done within 24 hours after the onset of symptoms in acute myocardial infarction. Because, after the first day, the total cholesterol and HDL cholesterol value will be decreased (HDL value decreased more than that of total cholesterol). True value will be obtained only after 8 weeks. Evidences supporting that cholesterol lowering therapy improve endothelial function and thus reducing risk of thrombus formation.

IMAGING STUDIES

CHEST X RAY

Chest X ray taken after initial stabilization of the patient may show degree of pulmonary congestion, severity of cardiomegaly. According to the findings, we can stratify the patients who are having high risk of mortality after the acute event. In right ventricular myocardial infarction, it shows clear lung field in earlier period, right atrial and right ventricular dilatation. It will also show the findings of chronic obstructive pulmonary diseases like barrel chest, prominent pulmonary artery which may be secondary to the pulmonary hypertension, which may be the cause of right ventricular myocardial infarction due to right ventricular hypertension. The other causes of chest pain can be studied by x ray chest.

ECHOCARDIOGRAPHY

The echocardiographic findings in right ventricular myocardial infarction includes the following [18]

1. Dilatation of right ventricle along with reduced dimension of left ventricle

2. Akinesia or dyskinesia involving right ventricular wall-indicates haemodynamically significant right ventricular myocardial infarction by identifying reduced motion of right atrioventricular groove due to reduction in longitudinal contraction of right ventricle.

3. Septal curvature reversal due to transeptal pressure reversal caused by increase in right ventricular end diastolic pressure.

4. Right atrial enlargement.

5. Bowing of inter atrial septum indicates associated atrial infarction.

6. Doppler echocardiography will identify tricuspid regurgitation, ventricular septal defect, patent foramen ovale, premature opening of pulmonary artery due to non-complaint right ventricle.

7. Dilatation of right atrium.

8. Dilatation and reduced collapsibility

9. Papillary muscle rupture or dysfunction

10. Thrombus in right atrium and ventricle & aneurysm of right ventricle.

HAEMODYNAMIC STUDY

Intracardic pressure and waveform related to right atrial and right ventricular contraction can be assessed rapidly after the onset of right ventricular myocardial infarction, by invasive method like insertion of catheter into the pulmonary artery. The findings suggestive of right ventricular myocardial infarction are elevated right atrial pressure (>10 mmhg) and ratio of right atrial and pulmonary capillary wedge pressure >0.8 (normal value is <0.6).

In right ventricular myocardial infarction the pattern of waveform depends upon atrial function.

In normal right atrial function prominent A wave and X descent, Y descent will be seen (W pattern). In atrial fibrillation, depressed A wave, diminished X and Y descent will be seen due to loss of right atrial function (M pattern).

In right ventricular waveform, broad and sluggish pattern due to reduced peak systolic pressure will be seen. Right ventricular diastolic dysfunction is indicated by increased right ventricular end diastolic pressure, equalization of diastolic pressure, right ventricular Dip and plateau pattern or square root pattern. This pressure equalization may be due to increased intra pericardial pressure caused by dilatation of right ventricle. [19,]

CARDIAC MAGNETIC RESONANCE IMAGING

By using gadolinium enhancement and T2 weighing imaging, this technique is used to analyze cardiac wall edema and regional wall motion abnormality.

RADIONUCLEOTIDE IMAGING

Radionucleotide angiography gives information about ejection fraction of right ventricle. But by knowing only ejection fraction we cannot diagnose right ventricular myocardial infarction with high accuracy. By combining wall motion abnormality with ejection fraction, the sensitivity (92%) and specificity (82%) increases in diagnosing haemodynamically

significant RVMI by this method. By technetium 99 pyrophosphate scintigraphy, the sensitivity to diagnose haemodynamically important right ventricular myocardial infarction is only 25%. Moreover we can't use this method in acute settings, and can be used only after initial stabilization.

CRITERIA TO DIAGNOSE RIGHT VENTRICULAR MYOCARDIAL INFARCTION [20]

ST elevation in right sided chest leads (V4R) >1mm

Right atrial pressure >10 mmhg or ratio of right atrial and pulmonary capillary wedge pressure >0.8

Right atrial waveform showing prominent A wave and X descent

Echocardiography or cardiac magnetic resonance imaging showing right ventricular asynergy.

MANAGEMENT

Early diagnosis of the right ventricular myocardial infarction is vital to the successful management of the patient because of following reasons.

Many acute clinical conditions resemble right ventricular myocardial infarction clinically (e.g. acute pulmonary embolism).

Electrocardiographic pattern of right ventricular myocardial infarction have broad differential diagnosis (e.g. pseudo anterolateral myocardial infarction).

Electrocardiographic features of right ventricular myocardial infarction has transient in nature (disappear within 12-24 hours)

Treatment profile of right ventricular myocardial infarction is different from other myocardial infarction.

Delaying treatment has high mortality. Otherwise early treatment has good prognosis in both short and long term outcome.

Initial stabilization of the patient

Vitals checking

Base line electrocardiography

Urgent intravenous line

Central venous line (if possible), for accurate hemodynamic monitoring

Continuous cardiac monitoring to detect arrhythmias

Nasal oxygen – All patients with right ventricular myocardial infarction should be given supplemental oxygen at the rate of 2-4 liter/minute through face mask or nasal prongs for period of at least 3 hours. Patients having pulmonary edema due to concomitant significant left ventricular dysfunction should have nasal oxygen for the period until disappearance of lung signs. Persistent pulmonary edema in spite of continuous oxygen therapy indicates patent foramen ovale due to elevated right atrial pressure or ventricular septal defect with right to left shunt of unsaturated venous blood

Analgesics

Patient with right ventricular myocardial infarction are frequently hypotensive and will have reduced preload to the left ventricle due its peculiar hemodynamic abnormalities (low output syndrome). So in this situation we should not use preload reducing drugs like morphine, which is otherwise drug of choice in reducing chest in comfort in other myocardial infarction. So, in acute right ventricular myocardial infarction, Inj.Pethidine should be used to alleviate chest discomfort. Intra muscular injections should be avoided.

Ant platelet drugs

All patients should be given T.Aspirin 325 mg stratum on admission, then 150 mg once daily thereafter. Aspirin irreversibly inhibits the cyclooxygenase enzyme thereby reduces Thromboxane A2 level and inhibits aggregation of platelets. In patients allergy to the Aspirin or contraindication to Aspirin, T.Clopidogrel a thienopyridine, 300 mg stratum, then 75 mg thereafter given in the absence of contra indications.

Nitroglycerine

In right ventricular myocardial infarction nitrates are generally contraindicated due to low output cardiac failure and hypotension. Nitrates due to its vasodilating nature of action, particularly of infarct related artery, reduces myocardial oxygen demand and increases oxygen supply. But in

the presence of significant left ventricular myocardial infarction and absence of hypotension, nitroglycerine can be safely used to reduce left ventricular after load and to increase cardiac output.

Bea blocker

Right ventricle has high sensitivity to the drugs which has action on the beta receptors particularly at the time of ischemia [21]. Because hypotension and bradycardia are the most common clinical complications of right ventricular myocardial infarction, using beta blocker will aggravate the already present hypotension and bradycardia. So beta blockers are generally contraindicated in right ventricular myocardial infarction.

PRELOAD OPTIMIZATION[22]

This part of treatment is the most important in the management of right ventricular myocardial infarction. Preload optimization done by volume loading with isotonic normal saline (0.9%). To start volume loading, initial assessment of the patient with regard to volume status is most important. Most of the patient of right ventricular myocardial infarction present with hypotension and clear lung field. These patients will benefit by volume loading. The main haemodynamic abnormality in patients with right ventricular myocardial infarction is under filling of left ventricle which is due to reduced right ventricular stroke volume and reduced volume size of left ventricle created by pushing of interventricular

septum towards left ventricle by dilated right ventricle and restraining effect of increased intra pericardial pressure. So by volume loading we can increase the filling pressure of right ventricle. According to Frank starling's law, stretching of right ventricular myocardial fiber, facilitates contraction of right ventricular myocardium and reverses all these abnormality. Not all patients are benefited by volume loading with isotonic saline. This indicates the role of restraining effect of increased intra pericardial pressure on the left ventricular filling and contribution of atrial contraction for right ventricular filling. Paramount importance is in avoiding preload reducing drugs like morphine, nitrates, diuretics and beta blockers. Optimum volume loading can be judged clinically by normalization of blood pressure and haemodynamic monitoring .In patients not responsive to initial volume loading, further volume challenge can be done with haemodynamic monitoring. If patients are not responsive to about 2 litres of normal saline or if central venous pressure does not rising to >15 mmHg & pulmonary capillary wedge pressure is <20 mmHg, further volume loading is not useful. In fact, further volume loading is deleterious to the patient by aggravating dilatation of the already failing, dilated right ventricle and thus increases chances of acute pulmonary edema. In right ventricular myocardial infarction associated with significant left ventricular myocardial infarction, intravenous nitroglycerine (in the absence of

hypotension) should be used to promote the left ventricular cardiac output, by reducing after load. In whom, cardiac output still does not increases, intravenous Dobutamine with or without intravenous dopamine should be used. As stated previously, ischemic right ventricular myocardium has high sensitivity to drugs acting on beta receptors, Dobutamine increases right ventricular contraction, particularly of interventricular septum. Inter ventricular septum contracts in a piston like manner, contracts towards right ventricular cavity. Thus increases right ventricular outflow and also increases left ventricular filling pressure. If cardiac output still does not improve then assist devices such as intra aortic balloon counter pulsation pump can be used.

RHYTHM OPTIMIZATION [23]

As right ventricular myocardial infarction commonly has rhythm abnormalities, control of cardiac rhythm is important in the management of right ventricular myocardial infarction. The following arrhythmias are commonly associated with right ventricular myocardial infarction. These are sinus bradycardia, atrial fibrillation, ectopic beats, and all degrees of atrio ventricular block, intra ventricular conduction defects, ventricular fibrillation and ventricular tachycardia. These arrhythmias further compromise the cardiac output, by causing atrio ventricular desynchronization

Brady arrhythmias usually respond to intravenous Atropine along with reperfusion therapy. If there is no response to intravenous atropine then temporary pacing may be required. Most of the patients will improve with this mode of therapy. A few patients will require permanent pacemaker therapy.

REPERFUSION THERAPY [24]

In all acute myocardial infarction patients, reperfusion therapy should be started as soon as possible after assessing the indication and contra indications. Reperfusion treatment improves both short and long term outcomes by reducing infarct size. It can be done by using drugs, known as fibrinolytic drugs or mechanical intervention Thrombolysis is indicated in all patients who presented within 12 hours of onset of myocardial infarction, and who have no contra indications.

Drugs used for thrombolysis

All these drugs convert plasminogen to plasmin, which degrades fibrin an important component of thrombosis of infarct related artery in all ST elevation myocardial infarction.

Streptokinase - intravenously-1.5 million units in 100 ml of normal saline over one hour. It is the most commonly used fibrinolytic agent in developing countries like India. It is non-fibrin specific. Side effects include hypotension, bleeding complication and allergic reactions.

Hypotensions (1-2%) especially occur on rapid infusion. So, if hypotension occurs, it is managed by temporarily slowing infusion or intravenous saline infusion and elevating the foot end. Allergic reactions occur if the patient has previous exposure to streptokinase (prior 5days to up to 2years) due to the development of antibodies.

Alteplase 15 mg intra venous bolus. Then 0.75 mg/kg intravenous infusion over 30 minutes. Then 0.5 mg/kg intravenous infusion over one hour period. Total dose should not exceed 100 mg. Alteplase is recombinant tissue plasminogen activator. Its fibrin specificity is only at modest level. It has small increase in the bleeding complication when compared to streptokinase.

Reteplase -given as two bolus intravenous doses as 10 units at 30 minutes interval. It is the deletion mutant of Alteplase. It is less fibrin specific than Alteplase. But it has longer duration of action when compared to Alteplase.

Tenectaplase- single intravenous bolus dose. It is the substitution mutation of Alteplase. It has high fibrin specificity and longer duration of action when compared to Alteplase. It is highly resistant to plasminogen activator inhibitor-1.It has lower bleeding tendency when compared to Alteplase. The dose is calculated on the basis of weight; 30mg for <60 kg; 35 mg for 60-70 kg; 40 mg for 70-80 kg; 45 mg for 80-90 kg;50 mg for >90 kg.

Glycoprotein 2b/3a inhibitor with reduced dose of thrombolytic agent – This method is not used extensively due to high incidence of bleeding complications. Moreover there is no increased mortality benefit when compared to fibrinolytic therapy alone.

CONTRAINDICATIONS FOR FIBRINOLYTIC THERAPY

Major contraindications

Known bleeding disorder

Aortic dissection

Gastrointestinal bleeding within preceding month

Major surgery or trauma with head injury within past 3 months.

Ischemic cerebrovascular accident within 6 months

Haemorrhagic cerebrovascular accident at any time.

Minor contra indications

Transient ischemic attack within past 6 months

Bleeding peptic ulcer

When patient is on anticoagulant therapy

Elderly age (>75 years)

Pregnant women or first week of post partum period

Hypertension (systolic blood pressure >180 mmHg in spite of treatment)

Infective endocarditis

Other co morbid conditions like cirrhosis, end stage renal disease

PERCUTANEOUS CORONARY INTERVENTIONS (PCI)

Percutaneous coronary intervention is one of the mode of reperfusion treatment. It is highly effective when patients with acute myocardial infarction presents within first few hours. It can also be done in patients with doubtful diagnosis, high risk of bleeding complications, when patient is in cardiogenic shock. When patients present more than 3 hours following acute myocardial infarction, the clot become more mature, so thrombolytic drugs do not easily penetrate and lyses the clot. In such a situation, percutaneous coronary intervention becomes a more useful procedure. Several types of percutaneous coronary interventions are there.

Primary percutaneous coronary intervention- this is when PCI done without previous attempt of thrombolysis.

Facilitated percutaneous coronary intervention- this is PCI done with previous glycoprotein 2b/ 3a inhibitor treatment.

Rescue percutaneous coronary intervention- this is PCI done after failed thrombolysis.

Elective percutaneous coronary intervention-this is PCI done for re-infarction following previous thrombolysis

The major disadvantages of percutaneous coronary intervention are its high cost, less availability, and requirement of highly skilled personnel support.

Successful reperfusion therapy is indicated by

1) Decrease in the chest discomfort

2) Resolution of ST segment elevation by at least 50% shown by electrocardiography taken 90 minutes following reperfusion therapy.

3) By angiography-**TIMI grading system** (Thrombolysis In acute Myocardial Infarction)-**Grade 0**-no flow of contrast material into the distal vascular bed of infarct related artery. **Grade 1**-flow of contrast into the infarct related artery is present but not into the distal vascular bed.**Grade 2**-flow of contrast into the distal vascular bed is present but at reduced flow rate compared to normal artery.**Grade 3**-floe of contrast material into the distal vascular bed at normal rate of flow.

Re infarction- indicated by recurrence of chest pain and ST segment elevation in electrocardiography. It is treated by rethrombolysis or angiography and percutaneous coronary intervention. Cardiac biomarkers like myoglobin and creatine kinase-MB has some role in diagnosing the reinfarction. In right ventricular myocardial infarction, reperfusion therapy if done earlier reduces infarct size, and thereby reduces in hospital mortality and complications like shock, complete heart block, and ventricular septal rupture. It also increases the effectiveness of volume loading in restoring the hemodynamic stability.

POST THROMBOLYTIC TREATMENT

Heparin- all patients with acute myocardial infarction should be started Inj. Heparin irrespective of thrombolysis (6 hours after thrombolysis if patient was thrombolysed) for up to 8 days of hospital stay as per 2008 ACC/AHA guidelines. Heparin therapy decreases infarct size by reducing thrombus formation and reduces risk of reinfarction. It also reduces risk of deep venous thrombosis due to immobilization of patients. But heparin therapy increases bleeding risk in thrombolysed patients.

Unfractionated heparin- 60 units/kg of unfractionated heparin followed by 12 units/kg up to maximum dose of 1000 units adjusted according to aPTT should be continued for 2 days following thrombolysis or until revascularization performed or up to 8 days of hospital stay period.

Low molecular weight heparin- two types of low molecular weight heparin are in use. These are enoxaparin and fondaparinux .

Enoxaparin –dose is 30 mg intravenous bolus, followed by 1mg /kg 12 hourly in <75 years age group. In >75 years of patients, the recommended dose is 0.75 mg /kg intravenously 12th hourly. It has slight increased risk of bleeding and significant decrease in the mortality and re-infarction when compared to conventional unfractionated heparin.

Fondaparinux- the dose is 2.5 mg once a day ,subcutaneously. Along with increased duration of action, it has marked reduction in death rate and

slight increase in bleeding risk, when compared to unfractionated heparin. Fodaparinux not used in patients who are undergoing percutaneous coronary interventions due to risk of catheter thrombosis.

Lipid lowering therapy- all patients admitted with acute myocardial infarction should receive therapy with statins to improve their lipid abnormality. Studies showed that better lipid control improves endothelial function and thereby reducing risk of thrombosis.

Anxiolytic drugs- all patients admitted with acute coronary syndrome should receive anxiolytic treatment in the form of Alprazolam to ensure calm environment.

Antacid drugs- all patients with acute myocardial infarction should receive H2 blockers or proton pump inhibitors. Proton pump inhibitors like Omeprazole reduce the effectiveness of antiplatelet drugs like Clopidogrel.

Stool softeners- like Bisacodyl should be given to all patients admitted with acute myocardial infarction. Because on straining for defecation, increased intra abdominal pressure imparts resistant to cardiac output which is intolerated by infarct recovering myocardium. That may lead to sudden cardiac death during hospital stay. Moreover constipation is the side effect of drugs like beta blockers which is used in acute myocardial infarction.

COMPLICATIONS OF RIGHT VENTRICULAR MYOCARDIAL INFARCTION

Cardiogenic shock

Arrhythmias like sinus bradycardia, complete heart block, atrial fibrillation, ventricular tachycardia and ventricular fibrillation.

Rupture of ventricular septum

Patent foramen ovale

Rupture of papillary muscle

Rupture of free wall of right ventricle and cardiac tamponade.

Pericarditis, aneurysm of right ventricle, pulmonary, thromboembolism.

MATERIALS AND METHODS

PLACE OF STUDY: Intensive Coronary Care Unit

Tirunelveli government medical college hospital,

Tirunelveli.

PERIOD OF STUDY: From october 2011 to november 2012.

STUDY DESIGN : Prospective Study

Ethical clearance : Obtained

Inclusion Criteria :

AGE : >20 years

SEX : male and female

ECG : Patients with ST Segment elevation in V4R

Exclusion Criteria:

Patients with congenital and other acquired heart diseases.

Patients with previous myocardial infarction

Patients with other serious co morbid illness like renal &liver failure.

PROTOCOL OF THE STUDY

Clinical history

Cljnical examination

Electrocardiogram

Echocardiogram

Clinical history

Age

Sex

Occupation

Socio-economic status

Admission time

Time of onset of symptoms prior to admission

Presenting history

Analysis of risk factors done with regards to

Body mass index

Diabetes Mellitus

Hypertension

Dyslipidimia

History of smoking

Family H/o Coronary artery disease (includes sudden Cardiac Death)

CLINICAL EXAMINATION

General examination

Pulse

Blood pressure

Respiratory rate

Temperature

SpO2-measurement of oxygen saturation by pulse oximetry

Jugular venous pulse

Kussmaul's sign

Cardiovascular examination

Risk stratification

TREATMENT

Bed rest

Nasal oxygen

Intravenous fluids

Narcotics for the control of chest pain

T. Aspirin 325mg stat

T. Clopidogrel 300mg stat

(Beta blockers, Nitrates, Diuretics, Morphine, and Enalapril- avoided if patient is in hypotension)

T. Alprazolam

T. Bisacodyl

Intra venous fluids – NS 100 ml/hr- if patient is having hypotension

If there is no response, then inotropic agents added (dopamine and/or dobutamine)

Reperfusion therapy

Inj. Streptokinase 1.5 million units in 100 ml of NS given over one hour with continuous cardiac monitoring to all patients with AMI who met the criteria for pharmacologic reperfusion therapy after ruling out the contraindications. Then patients were reassessed after 90 minutes of reperfusion therapy, for successful reperfusion therapy.

Reassesment done by

Clinical features – decrease in chest discomfort

ECG-resolving of ST elevation by 50% or more

All patients were continuously monitored to foresee the electrical and mechanical complications, re infarction, cardiac failure and treated as such.

INVESTIGATIONS

Urine –albumin, sugar, deposits

Blood -sugar (FBS, PPBS- in diabetic patients)

Urea

Serum creatinine

Serum electrolytes

Complete haemogram

Lipid profile

Troponin –T assay (By strip method)

Chest X ray

ECG- On admission

90 minutes after thrombolysis

Three times thereafter and

Whenever necessary

Echocardiography

(All the patients were obtained proper consent for the study)

OBSERVATION AND RESULTS

OBSERVATION OF CLINICAL FEATURES

AGE AND GENDER

Among 60 patients studied, 37 were male, 23 were female.

Serial no.	Gender	No. of patients	Percentage
1	Male	37	61.66
2	Female	23	38.33
Total		60	99.99

Sl.no.	Age	Male	%	Female	%	Total	%
1	21-30	3	5	0	0	3	5
2	31-40	5	8.33	0	0	5	8.33
3	41-50	21	35	3	5	24	40
4	51-60	6	10	12	20	18	30
5	61-70	2	3.33	8	13.33	10	16.66
Total		37	61.66	23	38.33	60	99.99

Lowest age found in this study was 28 years

Highest age found in this study was 70 years

Highest numbers of patients were found to be within the age group of 41-50 years in male and 51- 60 years in Female

Age	Male		Female	
	N	%	N	%
<50	29	78.4	3	13
>50	8	21.6	20	87
41-50	21	56.8	3	8.1

$\chi^2 = 21.981$ $df = 1$ $p < 0.001$

OCCUPATION

Among the manual workers, patients included were farmers, porters, rickshawpullers, etc. Skilled laborers included were carpenters, masons, etc., Professionals included were doctors, software engineers, teachers, auditors, etc.,

Patients having sedentary life included were office clerks, shop owners, etc., Of the patients, manual workers were the highest in number and professionals were the least in number.

Occupation	Number of patients			
	Male	Percentage	Female	Percentage
Manual workers	25	67.6	3	8.1
Skilled laborers	5	13.5	2	5.4
Professionals	3	8.1	2	5.4
Sedentary life	4	10.8	3	8.1
House wives	0	0.0	13	35.1
Total	37	100.0	23	62.2

RURAL AND URBAN VARIATION

Serial no.	Locality	No. Of patients	Percentage
1	Urban	15	25
2	Rural	45	75
Total		60	100

Place of residence	Male		Female	
	N	%	N	%
Rural	30	81.1	15	40.5
Urban	7	18.9	8	21.6
Total	37	100	23	100.0

$\chi^2 = 1.904$ $df = 1$ $p = 0.168$

Most of the patients studied here were from rural areas. They constituted by about 75 %. The patients from urban area constituted by about only 25%.

TIME OF ONSET OF SYMPTOMS

Time	12.01a.m-4.00 a.m	4.01a.m-8.00 a.m	8.01a.m-12.00 p.m	12.01a.m-4.00p.m	4.01 p.m-8.00p.m	8.01p.m 12.00a.m
Pts.	12	16	12	8	6	6
%	20	26	20	13.33	10	10

Time	12.01a.m-4.00a.m	4.01a.m 8.00a.m	8.01a.m 12.00p.m	12.01a.m 4.00p.m	4.01p.m 8.00p.m	8.01p.m 12.00a.m
Diabetics	5(8.33%)	7(11.66%)	5(8.33%)	7(11.66%)	5(8.33%)	4(6.66%)
Non-DM	7(11.66%)	9(15%)	7(11.66%)	1(1.66%)	1(1.66%)	2(3.33%)

Time	Non-diabetics		Diabetics	
12.01 a.m- 12.00 p.m	23	85.2%	17	51.5%
12.01 p.m-12.00 a.m	4	14.8%	16	48.5%

$\chi^2 = 7.576$ $df = 1$ $p = 0.006$

In this study, significant number of non-diabetic patients had onset of symptoms in the period between 12.01 a.m. - to 12.00 p.m., when compared to diabetic patients.

The association between diabetic status and time of onset of symptom was significant (p value<0.006)

TIME INTERVAL BETWEEN ONSET OF SYMPTOMS AND ADMISSION

Time Interval	<4hours	4-8 hours	8-12 hrs.	12-16hours	16-24hours
Male	7(11.66%)	14(23.33%)	9(15%)	5(8.33%)	2(3.33%)
Female	2 (3.33%)	4(6.66%)	6(10%)	7(11.66%)	4(6.66%)
Total	9 (15%)	18(30%)	15(25%)	12(20%)	6(10%)

Most of male patients presented within 12 hours. Of these, highest number of patients presented within 8 hours. Most of female patients presented at about 12 hours of onset of symptoms. Highest numbers of female patients were within the period of 12-16 hours.

Duration between symptoms onset and admission	<12hours	>12hours
Number of male patients	30(71.43%)	7(28.57%)
Number of female patients	12(38.89%)	11(61.11%)
Total number of patients	42(70%)	18(30%)

OBSERVATION OF SYMPTOMATOLOGY

Among 60 patients, studied 33 patients had typical symptoms of acute myocardial infarction. Remaining 27 patients had atypical symptoms. Among patients who had atypical symptoms, most of the patients (33.33%) were female. Whereas, in male patients 50 % had typical symptoms and 11.6% of patients had atypical symptoms. Most of the patients who had atypical symptoms were elderly and diabetics

Symptoms	No. Of patients	%	Male	%	Female	%
Typical symptoms	33	55	30	50	3	5
Atypical symptoms	27	45	7	11.66	20	33.33
Total	60	100	37	66.66	23	38.33

Atypical symptoms studied were giddiness, G.I.T. symptoms, syncope & dyspnoea.

	Male patients		Female patients		Total no. of patients	
Typical symptoms	30	81.1%	3	8.1%	33	55%
Atypical symptoms	7	18.9%	20	54.1%	27	45%
Total	37	100	23	100	60	100%

$\chi^2 = 26.528$ $df = 1$ $p < 0.001$

So, in this study significant number of female patients had atypical symptoms when compared to male patients. (p value <0.001)

OBSERVATION OF RISK FACTOR

Sl. no	Risk factors	Male	%	Female	%	Total	%
1.	Smoking	30	50	0	0	30	50
2.	Obesity	8	13.33	12	20	20	33.33
3.	Diabetes	16	26.66	17	28.33	33	55
4.	Hypertension	11	18.33	6	10	17	28.33
5.	Family h/o CAD	3	5	5	8.33	8	13.33

In this study, most common risk factor in male was smoking. In female, most common risk factor was DM. As a whole, DM was the

leading risk factor. Among these risk factors, family history of coronary artery disease was the least common.

OBSERVATION OF CLINICAL SIGNS

Although hypotension, clear lung field and elevated jugular venous pulse make the cardinal clinical signs of RVMI, not all the patients in this study had all of these three clinical signs. The most common clinical sign among the patients in this study was bradycardia. It was about 73%. The next most common sign was hypotension, which was about 68%. The elevation of JVP was noted in 77 % of these patients. Only 20 patients of this study were without pulmonary crepitations.

Sl. no.	Clinical signs	Total no. of patients	Percentage
1.	Hypotension	41	68.33
2.	Bradycardia	44	73.33
3.	JVP elevation	46	76.66
4.	Basal crepitations	38	63.33

JVP ANALYSIS -Among 60 patients of RVMI, 46 patients had elevated JVP. Of these, 15 patients had prominent A wave and X descent in JVP waveform .All those patients with prominent A wave and X descent in JVP presented with features of shock, which was not responding to volume loading with isotonic saline and inotropic therapy with doputamine. So, in

this study there was significant association between the prominent A wave, X descent in JVP and shock.

Shock	Prominent A wave and X descent in JVP				Total	
	Present		Absent			
	No. of pts.	%	No. of pts.	%	No. of pts.	%
Present	15	75.0	2	7.7	17	37.0
Absent	5	25.0	24	92.3	29	63.0
Total	20	100.0	26	100.0	46	100.0

$\chi^2 = 21.981$ $df = 1$ $p < 0.001$

OBSERVATION OF ELECTROCARDIOGRAPHIC FEATURES

In this study, none of the patients were admitted with isolated right ventricular myocardial infarction. Most of the RVMI patients had IWMI or IWMI and PWMI. Patients with RVMI and IWMI constituted 46.66%. Patients with RVMI, IWMI and PWMI constituted 48.33%. Only 5% of patients had AWMI along with IWMI.

Sl. no.	Pattern of MI	No. of patients	Percentage
1.	Isolated RVMI	0	0
2.	RVMI + IWMI	28	46.66
3.	RVMI+IWMI+PWMI	29	48.33
4.	RVMI+IWMI+AWMI	3	5

ST ELEVATION IN RIGHT SIDED CHEST LEADS (V4R)

ST elevation in right sided chest leads(V4R) was analyzed .All the patients had ST elevation in right sided chest leads. Of these, 22 patients had ST elevation >2mm and 38 patients had ST elevation < 2mm.All those patients who had ST elevation in V4R >2mm were significantly associated with complications, like cardiogenic shock, complete heart block and ventricular tachycardia/ ventricular fibrillation. These complications were less evident in those patients who had ST segment elevation (V4R) by <2mm

Comparisons of ST elevation in V4R lead according to complication

	Number of patients	Mean	SD	F	P value
No complication	35	1.4	.3	31.164	<0.001
Shock	17	2.2	.3		
CCB	4	2.2	.2		
VT/VF	4	2.1	.4		
Total	60	1.8	.5		

OBSERVATIONS OF ECHOCARDIOGRAPHIC FEATURES

All of the studied patients were underwent echocardiography after emergency treatment and haemodynamic stabilization. The echocardiography was analyzed in terms of right ventricular dyskinesia,

right ventricular dilatation, paradoxical interventricular septal movement and other previously mentioned abnormalities.

Sl. no.	ECHO features	No. of patients	%
1.	RV dyskinesia	22	36.66
2.	RV dilatation	12	20
3.	Paradoxical IV septal movement	8	13.33
4.	Right atrial dilatation	6	10
5.	VSR,PFO, papillary muscle rupture	0	0
6.	Anterior wall infarction	3	5
7.	Significant TR	7	11.66

Right ventricular dyskinesia was present in 36.66 % of patients of right ventricular myocardial infarction. Right ventricular dilatation was observed in about 20% of patients. Paradoxical septal motion towards left ventricular cavity was seen in 13.33% of patients with right ventricular myocardial infarction. Atrial dilatation was the least commonly seen feature among all patients of right ventricular myocardial infarction, which was about 10 %. Of the echocardiographic features observed, right ventricular dyskinesia ,right ventricular dilatation, paradoxical septal movement towards left ventricle and right atrial dilatation was consistently associated with

complications like cardiogenic shock, compete heart block, ventricular fibrillation/ ventricular tachycardia and atrial fibrillation.

Right ventricular dyskinesia was present in 22 out of 60 patients with right ventricular myocardial infarction. All the patients with right ventricular diskinesia were associated with complications like cardiogenic shock, complete heart block, ventricular tachycardia/ventricular fibrillation.

Right ventricular dilatation was present in 12 patients of right ventricular myocardial infarction. The occurrence of right ventricular dilatation was less, when compared to that of right ventricular dyskinesia ;(36.66% of RV dyskinesia vs. 20% of RV dilatation). All the patients with right ventricular dilatation had fatal complications like cardiogenic shock, complete heart block, ventricular tachycardia/ventricular fibrillation.

Paradoxical septal movement of inter ventricular septum towards left ventricular cavity was seen in 33.33% of patients with right ventricular myocardial infarction. Among the patients with paradoxical septal movement, most of the patients had features of cardiogenic shock, ventricular tachycardia/ventricular fibrillation and complete heart block.

Right atrial dilatation was seen in 6 patients of right ventricular myocardial infarction. All these patients who had right atrial dilation also had right ventricular dilatation, right ventricular dyskinesia and paradoxical septal movement towards left ventricular cavity. Only two of the patients

with right atrial dilatation had associated transient atrial fibrillation lasting for about 6 hours.

Significant tricuspid regurgitation was seen in seven out of the 60 right ventricular infarction patients. All the patients with significant tricuspid regurgitation, also had right ventricular dyskinesia, right ventricular dilatation, and paradoxical septal movement towards left ventricular cavity, and right atrial

dilatation.

All the patients with right ventricular infarction had inferior wall infarction. 29 out of 60 patients who had posterior wall infarction also had right ventricular MI and inferior wall infarction. Three of the patients who had anterior wall infarction also had right ventricle and infero-posterior wall infarction. None of the patients in this study had ventricular septal or free wall rupture, papillary muscle rupture and patent foramen of ovale.

ANALYSIS OF COMPLICATIONS

The complications observed in this study include cardiogenic shock, electrical complications like sinus bradycardia, all degree of atrio ventricular block, intra ventricular conduction defects, atrial fibrillation ventricular tachycardia, ventricular fibrillation and death. Among these complications the most frequently observed one was sinus bradycardia. The

least common of these complications seen in the patients with right ventricular myocardial infarction was atrial fibrillation.

Sl. no.	Type of complications	No. of patients	%
1.	Cardiogenic shock	17	28.33
2.	Electrical complications	48	80
3.	Mechanical complications	0	0
4.	Death	5	8.33

The electrical complications were seen in 80 % of the patients of RVMI. No one patient was noted to have mechanical complications like ventricular septal or free wall rupture, papillary muscle rupture, patent foramen of ovale.

Electrical complications- In this study, out of 60 patients of right ventricular myocardial complications, 48 patients were having electrical complications. Sinus bradycardia was seen in 44 patients of right ventricular myocardial infarction. The RBBB was present in 20 patients of total 60 right ventricular myocardial infarction. The intra ventricular conduction defect, left anterior hemi block was seen in 6 patients. The patients who had first degree atrio ventricular block accounted to be 24 out of 60 right ventricular myocardial infarction patients. Second degree atrio ventricular block was present in seven patients. The most severe one,

complete heart block was present in only four patients. The fatal ventricular tachycardia/ventricular fibrillation were present in four out 60 patients with right ventricular myocardial infarction. The least common arrhythmia seen in this study was atrial fibrillation, which was seen in two patients. Atrial fibrillation here was transient in nature, last for around six hours.

Sl. no.	Arrhythmia	Total no.of patients	Percentage
1	Sinus bradycardia	44	73.33
2	First degree AV block	24	40
3	Second degree AV block	7	11.66
4	Complete heart block	4	6.66
5	RBBB	20	33.33
6	LAHB	6	10
7	Atrial fibrillation	2	3.33
8	VT/VF	4	6.66

MANAGEMENT

Of the 60 study patients, one patient died within 5 minutes of receiving time, immediately after taking electrocardiogram and checking of vital parameters. That patient presented with features of severe cardiogenic shock with impalpable pulse, immeasurable blood pressure, ST segment elevation in inferior wall leads, posterior wall leads, anterior wall leads in

addition to ST segment elevation in right sided chest leads (ST elevation in V4R <2mm). All the other patients received emergency measures for the stabilization while simultaneously taking baseline electrocardiography. Among these 59 patients, 36 patients were thrombolysed with Inj. Streptokinase 1.5 million units in 100 ml of normal saline through intravenous infusion over the period of one hour, after considering the indications and contraindications for thrombolysis. Irrespective of the thrombolysis, all the patients were given Inj. heparin (low molecular weight heparin) subcutaneously after considering contraindications. All the thrombolysed patients were given Heparin Injection 6 hours after thrombolysis. All the patients were taken electrocardiography 90 minutes after thrombolysis, then three times a day and whenever necessary. All the 59 patients subjected to echocardiography study after haemodynamic stabilization and thrombolysis (in patients who had indications for thrombolysis)

COURSE OF THE PATIENTS DURING HOSPITAL STAY PERIOD

Except one patient, who died immediately on arrival, all the other 59 patients improved following initial emergency stabilization measures. All the patients who were thrombolysed improved both symptomatically and haemodynamically. The rate of occurance of complications like cardiogenic shock, complete heart block, VT/VF was less when compared

to non thrombolysed patients. Significant number of non thrombolysed patients also improved both clinically and haemodynamically. But the rate of occurrence of complications including death was high among these non thrombolysed patients. Three of the non thrombolysed patients died, whereas only one of the patients died among thrombolysed patients. All the other patients survived hospital stay period, although some of them continuously had few complications and referred for higher centre for further management.

Total no.of RVMI pts.	Thrombolysed	%	Non-thrombolysed	%
60	36	60	24	40

Sl. no.	Complications	Inj. S.K. given	%	Inj. S.K. not given	%
1	Cardiogenic shock	6	16.67	11	45.83
2	Complete heart block	1	2.77	3	12.5
3	VT/VF	2	5.55	2	8.33
4	Death	1	2.77	4	16.66

MORTALITY

Out of 60 study patients five patients were died. One patient died immediately on arrival while the urgent resuscitative measures were taken. The other patients died during the hospital stay period.

DISCUSSION

ANALYSIS OF CLINICAL FEATURES

AGE AND GENDER ANALYSIS

In our study men were predominantly affected than women .Affected men constituted 61.66%, women constituted 38.33%. This data parallels with data observed in many Studies. Azhar et al found that the incidence of RVMI was about 89% in male and 91% in female [25]

REASON

In most of Indian families, male are earners and female are usually house wives. So the male are exposed to accidents, violence and stress at higher level than female. Although psychological stress is commonly present in female, male are additionally having the habit of smoking which is the more prevalent and most consistently associated risk factor for AMI in male in India, which is discussed below. In Indian context prevalence of smoking among female is very less when compared to western countries.

AGE

In our study the incidence of RVMI in male was higher within 40-50 years of age group than other age groups; Most of female were within 50-60 years of age group. All the female patients with RVMI were more than 40 years of age. Whereas 8 of male patients were under the age of 40 years. In this study, about 78.4% of male patients were under the age of 50 years. In

contrast, 87 % of female patients were above the 50years of age .In this study, the association of RVMI in male and female patients with regards to age group was significant (p value= <0.001). Anyway, the age incidence of RVMI of both sexes in this study was 10 years lower than that of many studies conducted in western countries In one study Azhar et al and others [9,25] found that the highest number of RVMI occurs within the age group of 37-70years (mean age-54 years).

REASON

This may be due to premature occurrence of CAD in Indian people. This prematurity of CAD in Indian context is due to high risk factor levels at younger age. In a study conducted in India at Calicut Medical College in Kerala, it was seen that 1st AMI in patients younger than 40 increased up to 20 times between 1971 and 1991. This inference was supported by various studies and also by INTER HEART-SOUTH ASIAN study[26] and SHARE study[27]. These studies showed Indian male and female are prone to develop CAD by 10 years earlier than peoples of other countries. This is consistent with our study. However in our study women are presented with MI 10 years later than men. This may be due to loss of protective effect of ovarian hormones (such as anti-atherogenic and vasodilatation) in the post menopausal age group , which was the predominant age group of RVMI presentation noted among women in our study. But in view of this concept,

use of hormone replacement therapy in the prevention of coronary heart disease in women is not accepted widely, due to adverse prothrombotic effect of estrogen in HRT. HERS Study (1998) showed that HRT has no protective effect on prognosis of postmenopausal women with coronary artery disease. WHI-Study (2002, 2004) showed that HRT has no beneficial effect on healthy post menopausal women in respect of CAD[28-31].

LOCALITY AND OCCUPATION ANALYSIS

In this study, most the of patients were manual workers (36.66%) who were actually farmers, porters, rickshaw-pullers, etc. Only three of the female patients were manual workers. Most of the female patients were housewives. Almost all of them came from nearby villages. This inference contrast from many studies conducted across various parts of India. These studies showed that in India the prevalence of CAD increased two-fold in rural areas and nine fold in urban areas increased.

REASON

The reason for more presentation of rural people in this study may be due to the following factors .Poor educational level, low socio-economic status, poor availability of health facilities and faculties, poor knowledge about CAD preventive measures. Studies showed that use of green leafy vegetables and fruits protect the individual from coronary heart disease. Although village peoples are having high availability of fresh vegetables,

poor knowledge about advantage of using vegetables and poor cooking practices prevent them from getting the benefit of vegetables. In Indian rural household, prolonged cooking of vegetables leads to loss of 90% of its folate content. A study conducted in India showed that there is inverse association between consumption of vegetables and occurrence of ACS[32,33,34]

One important observation in this study was the prevalent of bidi smoking among rural peoples. Bidis are prepared from temburni leaf by hand rolling. These are prepared in most of the villages in Tirunelveli district. These are unfiltered and more atherogenic one, available at low cost, when compared to cigarettes. Because of its low cost and easy availability in rural areas, it may be the one of important factors contributing to more prevalence of RVMI among rural people in this study. T Rastogi et al found importance of bidi smoking as a risk factor for the development CAD in Indian male[35].As this study was conducted in regional government general hospital, which is the more easily available health referral system for poor rural people, the true prevalence of RVMI in rural and urban people could not be assessed.

RISK FACTOR ANALYSIS

In our study smoking was the most common risk factor for male. Whereas DM were the most common risk factor for female. As a whole, DM was

the leading risk factor. This observation was supported by number of studies conducted towards the prevalence of risk factor in Indian people. INTERHEART STUDY –SOUTH ASIA[26] showed that eight risk factors were consistently associated with Indian people with CAD. These are abnormal lipids, smoking, HTN, DM, truncal obesity, psychosocial factors, low fruit and vegetable intake, and lack of physical activity .It also Showed that these are responsible for 89% of the acute coronary syndromes in Indians. Among these risk factors, smoking comes second to abnormal lipids. Abnormal lipids defined in the INTERHEART-South Asia study was Apo-lipoprotein B-100 and ratio of Apo-lipoprotein B-100 and A-1 .Both of these were shown to be elevated in most of Indian peoples with CAD. In our study, tests for these types of abnormal lipids were not done, due to the reasons of non-availability and high cost. In our study people, bidi smoking was the leading risk factor due to its low cost and easy availability as discussed previously. With regard to prevalence of risk factors in female with RVMI, DM was the leading risk factor in our study people. This is because most of the women participated in our study were house wives and they were also more number of obese than men in this study. So, the occurrence of DM in our obese female patient as a part of metabolic syndrome (syndrome X), will explain the highest prevalence of DM in female patients in our study .Moreover Sullivan AK et al and

Douglas PS et al concluded in a study that DM has stronger association in female patients with CAD .In our study DM was the leading risk factor for RVMI as a whole. This correlates with the fact that India is known as the diabetes capital of the world; because in India diabetes is 2 to 4 times more common than rest of world. DM was the second common risk factor in our male patients whose are manual workers and mostly thin individuals when compared to female patients. This high incidence of DM in non -obese thin patients is supported by the observations in INTERHEART STUDY-SOUTH ASIA .This study showed that diabetes occurs earlier (10 to 15 years) and at a lower body weight (9.1 to 13.6 kg) in Indians than other people. Regarding prevalence of HTN, this comes next to the DM, & smoking in our study. CURES cohort by Mohan et al pointed out that in Chennai every fifth individual is a hypertensive, which equals or even override the DM prevalence.[36,37,38,39] Regarding novel risk factors, INTERHEART STUDY-South Asia stated that other than Apo-lipoprotein B-100 and B-100/A-1 ratio, other novel risk factors like homocysteine in Indians are of little value. The non-coronary risk factors contributing for Right ventricular myocardial infarction are right ventricular hypertrophy of any cause and iatrogenic occlusion of right coronary artery or its branches. Normally low O2 requirement makes right ventricle less amenable to development of infarction. This is due to its thin walled structure, less work

load of right ventricle in pumping out of blood against low-resistant pulmonary vascular bed, and presence of rich collateral network system. But in the setting of right ventricular hypertrophy its O2 requirement increases. If RVH is associated with even haemodynamically insignificant atherosclerotic obstruction of RCA, risk of RVMI increases. RVH secondary to respiratory cause (i.e. COPD) is particularly important because it will cause both hypoxemia and increased demand due to RVH. Iatrogenic RVMI can occur in the setting of invasive cardiac intervention like PCI involving right coronary artery. Acikel et al & van der Bolt et al showed RVMI secondary to isolated acute occlusion of a large RV branch of the RCA following coronary balloon angioplasty in their study.

TIME OF ONSET OF SYMPTOMS

Among our study people, most of the patients had time of onset of symptoms between 12.01 A.M. to 08.00 A.M. Among these, most of the patients were non-diabetic. Most of the diabetic patients had symptoms onset during the rest of day. In the present study 85.2 % of the non diabetic patients and 51.5 % of diabetic patients had onset of RVMI symptoms during 12.01 A.M. -12.00 P.M. But 14.8% of non-diabetic and 48.5 % of diabetic patients had onset of symptoms during 12.01 P.M.-12.00 A.M. The association between the incidence of diabetes and loss of circadian rhythm seems to be significant in the present study.(p value is <0.006)

REASON

The reason for this loss of circadian variation was studied in detail in the past. Diabetic autonomic neuropathy due to long standing diabetes leads to attenuation of circadian rhythm of ACS. Abnormalities in the circadian rhythm of autonomic tone are the most acceptable explanation for this. This inference is supported by study done by

Hjalmarson et al and many others [40,41] .

TIME INTERVAL BETWEEN ONSET OF SYMPTOM AND ADMISSION

In our study, the time interval from onset of symptoms to admission vary considerably between male and female. Most of the male presented within 4-8 hours period. But most of female presented within 8-16 hours period. Of about 70 % of patients presented within 12 hours of onset of symptoms. Among these, male patients contributed to about 71.43 % and female patients contributed to about 28.57%. Only 30 % of patients presented more than 12 hours after onset of symptoms. Among these patients female contributed (61.11%) more than male (38.89%)

REASON

This late presentation in female may be due to higher prevalence of atypical symptoms in female. Atypical symptoms of AMI such as indigestion, giddiness, vomiting, dyspnoea, epigastric pain are more

common in women. In this study, most common symptom in female was indigestion. This type of symptom may lead to misinterpretation of RVMI as disease of GIT both by patient themselves and by the attending primary health care physician. This was shown by many studies previously. Stronger influence of DM in women with CAD and elderly nature of female patients in this study may add to this effect due to autonomic neuropathy. Social negligence of female may contribute to late presentations of female with acute myocardial infarction in some regions.[42]

ANALYSIS OF SYMPTOMATOLOGY

In this study, 55% of the patients (33 patients) had classical chest discomfort. Among these 50 % of male patients (30 patients) and 5 % of female patients (3 patients) accounted for typical chest discomfort of RVMI. An atypical symptom was observed in 45% of patients (27 patients).Of these 33.33% of female (20 in number) and 11.66% of male (7 in number) accounted for atypical symptoms like indigestion, epigastric pain and syncope. In this study, occurrence of atypical symptoms was noted more in the elderly patients than younger patient; diabetics more than non diabetics; female more than male. Here the association of typical and atypical symptoms among male and female patients respectively was significant (p value <0.001).This observations correlates with many studies which showed more prevalence of atypical symptoms among women.

REASON

Predominance of atypical symptoms in female in the present study correlates with previous studies. Most of RVMI associated with IWMI, which is more frequently associated with GIT symptoms due to increased diaphragmatic and vagal activities (BEZOLD-ZARISH REFLEX).

ANALYSIS OF SIGNS

In this study, around 68.33 % of patients had hypotension, 73.33% of patients had brdycardia, around 76.66% of patients had elevated JVP and 63.33% of patients had basal crepitations. Among 46 patients, who had elevated JVP, and 20 patients showed prominent A wave and X descent in the JVP waveforms. Of these 20 patients with prominent A wave and X descent in JVP wave form, 75 % of patients subsequently had cardiogenic shock. This association of haemodynamically significant RVMI with prominent A wave and X descent in JVP was significant (p value 0.<001). This correlates with many studies conducted in the past. In a study conducted by Dell Ialia LJ et al it was found that elevated JVP alone has 88 % sensitivity and 69% specificity in diagnosing haemodynamically significant RVMI . Goldstein et al found that prominent A wave and X desend was most significantly associated with severe right heart failure in RVMI patients who had intact right atrial function .Although hypotension, elevated JVP, clear lung field makes clinical triad of RVMI , it will occur

only in haemodynamically significant RV infarct as a features of right heart failure. This observation is similar to observation made in SHOCK trial .Among patients had this clinical triad, most of patients improved with fluid therapy. Rest of the cases, who did not improv with fluid therapy, who also had prominent A wave in JVP, and creptations in lung bases improved with dobutamine. This may be due to extensive right ventricular infarct or associated LV dysfunction. This observation points out the importance of identifying haemodynamically significant RVMI by careful observation of JVP as a simple bed side method to identify patients who require judicious use of morphine, nitrate, and beta blockers. This inference was similar to observation made in the SHOCK trial and Dell'' Italia LJ et al .In the present study 28.33% of patients had haemodynamically significant RVMI in the form of cardiogenic shock. Most of these patients improved with infusion of normal saline and intravenous infusion of Dobutamine in addition to the urgent thrombolysis. This observation correlates with many studies, which showed that only15-30% of patients of RVMI were haemodynamically compromised, even though >30% of RVMI patients occurred in IWMI patients.

ANALYSIS OF ECG FEATURES

In this study almost all the patients had ST elevation in RV4. Invariably all the patients were associated with IWMI and most of them, were associated

with PWMI also .None of the patients had isolated RVMI. The studies conducted in the past showed that isolated RVMI is very rare. The RVMI is usually seen in patients with infero-posterior wall MI. Studies showed that incidence of isolated RVMI is <3%, the incidence of IWMI with RVMI is >30%. In the present study 100 % of RVMI patients also had IWMI and 51.66% of RVMI patients had the involvement of posterior wall. Only 5 % were associated with AWMI .One of important features of ECG analysis was the degree of ST elevation in RV4. All the patients of RVMI had ST elevation >1mm in V4R. Of these about 36.7% of patients had ST elevation >2mm. Among these patients who had higher degree of ST elevation, most of patients also had clinical features of cardiogenic shock (68.2%). So from this study it is well known that amount of ST elevation in RV4 can be used to asses haemodynamically significant right ventricular myocardial infarction. This observation corresponds to conclusion made in a study by zehender et al[43]. Another feature of RVMI is its transient nature of ST elevation. In our study it was found that ST elevation in V4R lead persisted up to 2 days. In a study conducted by Fijewski TR et al and klein HO et al showed that persistence of ST segment elevation upto12- 24 hours.[14,15]

ARRYTHMIAS

In this study about 80 % of patients had electrical complications in the form of sinus bradycardia, all degrees of AV block, right bundle branch block,

left anterior hemiblock, atrial fibrillation and VT/VF. Right ventricular myocardial infarction increases the risk of arrhythmic complications and death among the patients with inferior wall myocardial infarction[23].In this study most common arrhythmia encountered was sinus bradycardia(73.33%). This may be due to increased activity in vagal afferent fibers. Vagal nerve fibers more commonly present in the inferior aspect of heart. So, IWMI patients are having more chances to develop bradyarrhythmias even without the presence of RVMI, due to the presence of Bezold- Jarisch reflex. In the present study, all the RVMI patients were associated with IWMI. This may be the reason why sinus bradycardia was the most common arrhythmia in the present study. In right ventricular myocardial infarction the development of bradycardia more complicates the already existing hemodynamic disturbances by producing atrio ventricular desynchronization and reducing the contribution of right atrial contraction to the right ventricular filling. In the present study, significant number of patients of sinus bradycardia improved with conservative therapy. Some cases who did not respond to conservative treatment, required temporary pacemaking and very few of the patients were in the need of permanent pacemaker .This observation correlates with many studies and a study conducted by Love jc et al, who showed similar improvement of sinus bradycardia following conservative management like

intravenous atropine and aminophilline.In the present study, complete heart block was seen in 6.66% of patients. All these four patients were associated with cardiogenic shock, ST segment elevation >2mm in V4R, echocardiographic features of right ventricular dyskinesia, right ventricular dilatation and paradoxical septal movement towards left ventricular cavity. Of patients had VT/VF, significant number of patients had shock, higher degree of ST segment elevation in V4R and echocardiographic features suggestive of severe right heart failure, like previously mentioned. In a study conducted by brugada et al, 48% of patients with right ventricular myocardial infarction developed complete heart block and most of the patients improved with intravenous injection of atropine following thrombolysis. In the present study also similar observations were noted. In this study, 75 % of patients with complete heart block improved with intravenous atropine following thrombolysis.In this study VT/VF occurred in 6.66% of the patients. Among these all the patients were associated with significant haemodynamic disturbances in the form of cardiogenic shock. Of these, about 75 % of the patients withVT/VF died, and only 25% patients survived. So VT/VF became the most common cause of the death in this study. This observation showed that these arrhythmic complications in RVMI likely to be present in severe cases of RVMI and these electrical complications itself worsens the already existing haemodynamic

disturbances. This inference correlates with many studies conducted towards complications of right ventricular myocardial infarction.

ANALYSIS OF ECHOCARDIOGRAPHIC FEATURES

In the present study, 36.66 % of RV diskinesia,20% of RV dilatation,13.33% of paradoxical inter ventricular septal movement towards left ventricular cavity, 10% of RA dilatation and bowing of inter atrial septum towards left atrium, 11.66% of significant tricuspid regurgitation and 5% of left ventricular involvement was noted among all RVMI patients. In the present study, most of the RVMI patients, who had cardiogenic shock,complete heart block and VT/VF, all sho had RV dyskinesia,RVdilatation and paradoxical IV septal movement towards interventricular cavity.So, this study showed that RV dyskinesia, RV dilatation and paradoxical movement of interventricular septum towards left ventricle are highly important echocardiographic features in detecting heamodynamically significant RVMI. This observation met with study conducted by Dell LJ et al[21].

RA dilatation and bowing of inter atrial septum towards left atrium was seen in 10% OF RVMI patients in this study. These two patients were seen to be associated with transient atrial fibrillation lasting about 6 hours with temporary worsening of heamodynamic parameters. This association of bowing of inter atrial septum in patients with atrial fibrillation was

significant and correlates with study conducted by Dell Italia LG et al and others.[44-50]

ANALYSIS OF TREATMENT AND OUTCOME

Among patients studied, 8.33% of patients died. In CORE trial it was found that RVMI increases the mortality, cardiogenic shock and arrhythmic complications by three fold in patients of IWMI, when compared to IWMI alone. In the present study, although more death (60%) occurred among non thrombolysed patients, considerable amount of death (40%) also occurred in thrombolysed patients. It indicates that thrombolysis is not well impressive in patients with RVMI, even though it reduces mortality, shock and arrhythmic complications among RVMI patients. This observation correlates with observations made by Bates ER et. al[44]. It also indicates that the preload optimization, inotropic treatment, rhythm optimization are more important in the management of RVMI patients. In a study, Gold stein et al found the importance of preload optimization and rhythm optimization. Dell Italia et al and Brooks et al found that the importance of inotropic support in RVMI patients. Our study correlates with observations made by Dell Italia et al and Brooks et al[21,45].

SUMMARY

1. RVMI occurs predominantly in male patients
2. Atypical symptoms are more common in female
3. None of the case presented with isolated RVMI
4. RVMI usually associated with IWMI
5. Severity of RVMI can be assessed by JVP analysis, ST elevation in V4R in ECG and by ECHO.
6. Smoking is the most common risk factor in male, diabetes in female and overall diabetes is the most common risk factor.
7. Most of the patients were from rural areas
8. Death rate not markedly differs between volume loading and thrombolysis
9. Volume loading therapy is almost equal to thrombolysis in RVMI, in terms of efficacy.
10. CAD has increased in incidence among lower socio economic people. It has the equal incidence among them, when compared to higher socio economic people.

CONCLUSION

1. The incidence of right ventricular myocardial infarction is increasing.
2. RVMI equally affects lower socio economic people.
3. Smoking now becomes the most common cause of RVMI in male..
4. Diabetes is the leading cause of RVMI in both male and female.
5. Isolated RVMI is very rarely occurs.
6. RVMI is usually associated with inferior wall myocardial infarction.
7. So, all patients with IWMI must have right sided chest leads (V4R), which is the most easily available means to diagnose RVMI at earlier period.
8. RVMI if diagnosed earlier has good prognosis.
9. If appropriate preventive measures taken, not only RVMI, the burden of coronary artery disease can be decreased in the community.

LIMITATIONS OF THE STUDY:

1. This study was conducted during the course of one year period only.
2. Here, the true prevalence of RVMI among the rural and urban people cannot be studied, as this study was conducted in govt. general hospital which is predominantly utilized by poor rural people.
3. Here, the tests for Novel risk factors were not done due to non availability and its high cost.

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ABBREVIATIONS

CVD-cardiovascular disease

CAD-coronary artery disease

MI-myocardial infarction

RA-right atrium

RV-right ventricle

LV-left ventricle

IVS-inter ventricular septum

RVMI-right ventricular myocardial infarction

IWMI-inferior wall myocardial infarction

PWMI-posterior wall myocardial infarction

AWMI-anterior wall myocardial infarction

RVH –right ventricular hypertrophy

COPD-chronic obstructive pulmonary disease.

CHB- complete heart block

VT/VF-ventricular tachycardia/ventricular fibrillation

AF-atrial fibrillation

ECG-electrocardiography

ECHO-echocardiography

VSR-ventricular septal rupture

PFO-patent foramen ovale

PCI-percutaneous coronary intervention.

PROFORMA

A STUDY ON CLINICAL PROFILE OF RIGHT VENTRICULAR MYOCARDIAL INFARCTION

Name-

Age -

Sex -

Occupation -

Address –

Date of admission –

Time of admission-

Presenting history

Time of onset of symptoms- 93

Past history-

DM

HTN

FAMILY H/O CAD

Personal history

Diet

Smoking

Alcohol

OC pills

Menstrual history

Drugs/history 94

Family history

DM

HTN

CAD

PVD

CVA

DYSLIPIDEMIA

General examination

General appearance

Attitude

Extremities 95

Anemia

Jaundice

Cyanosis

Clubbing

Pedal edema

Examination of vital signs

Pulse

Blood pressure

Respiratory rate

Temperature

Jugular venous pulse 96

Systemic examination

Cardio vascular system –

s1,s2

S3,s4

Murmur

Pericardial rub

Respiratory examination

Breath sounds

Crepitations

Investigations

Basic investigations

Electrocardiography

Echocardiography 97

APPENDIX

Chart – 1

Gender Distribution

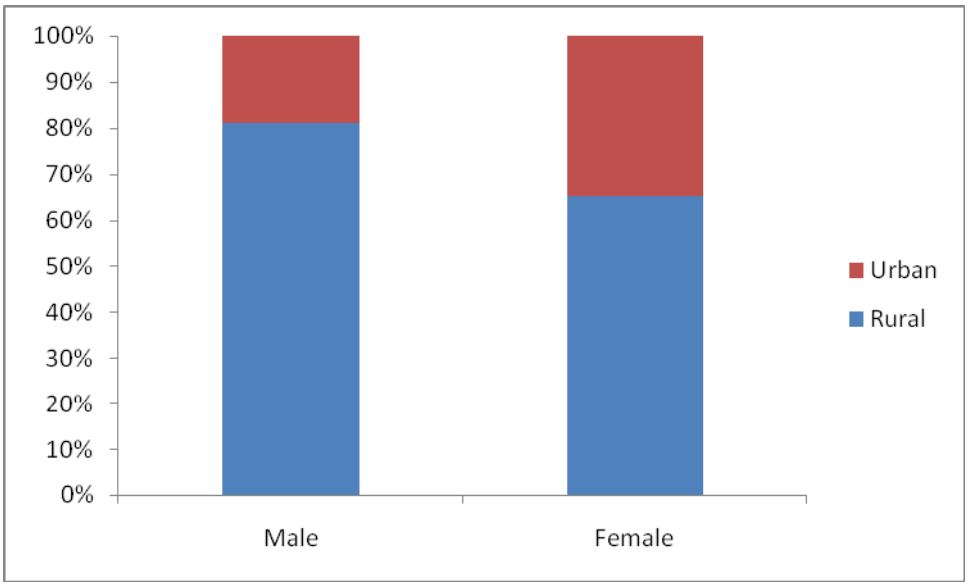


Chart - 2

ECHO – FINDINGS

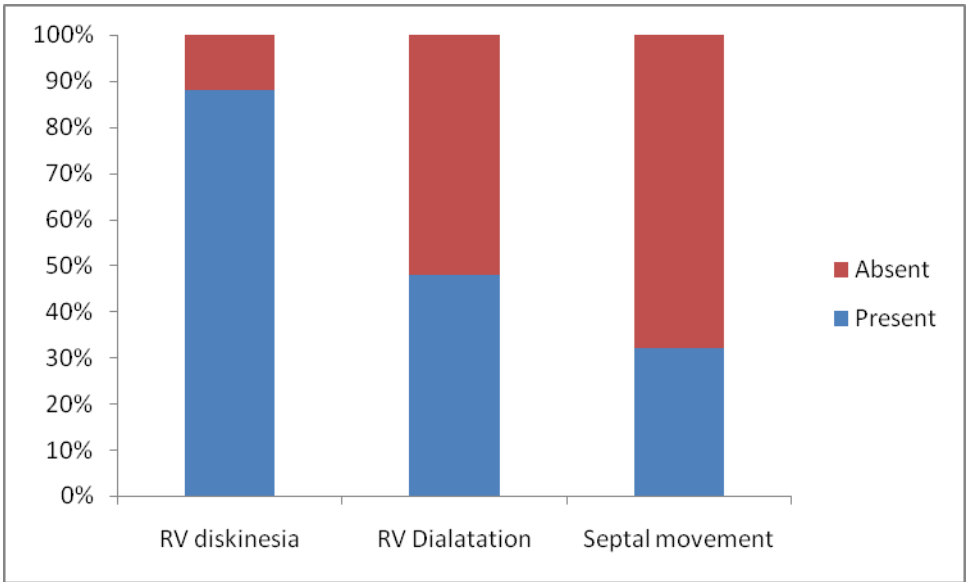


Chart - 3

TIME OF ONSET OF SYMPTOMS

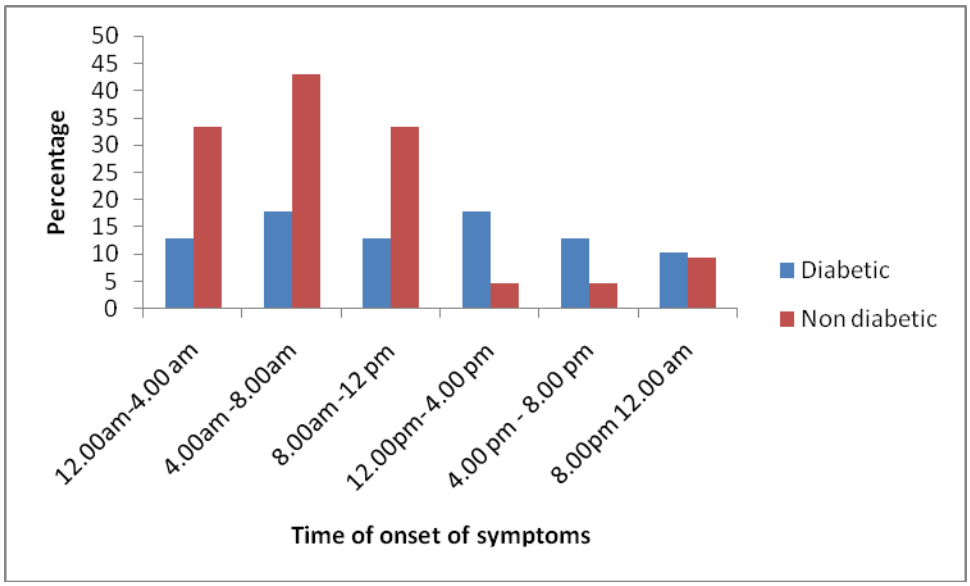


Chart - 4

ST ELEVATION IN V4 R LEAD

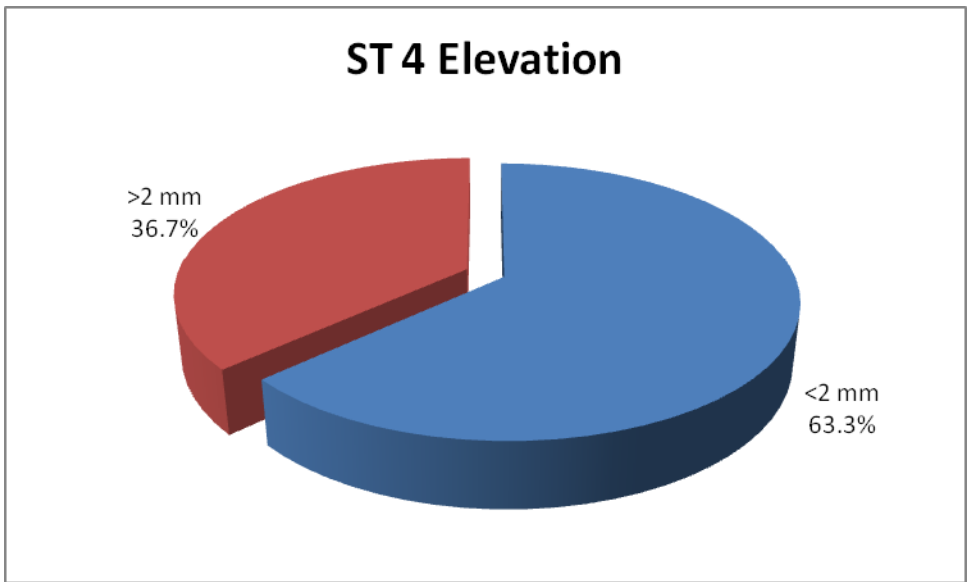


Chart - 5

ST ELEVATION V4R AND COMPLICATIONS

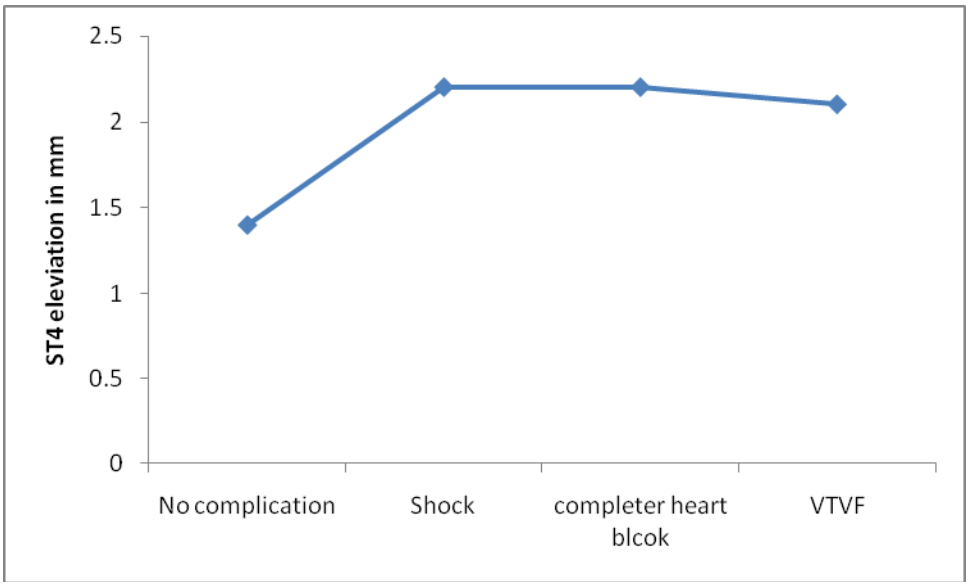
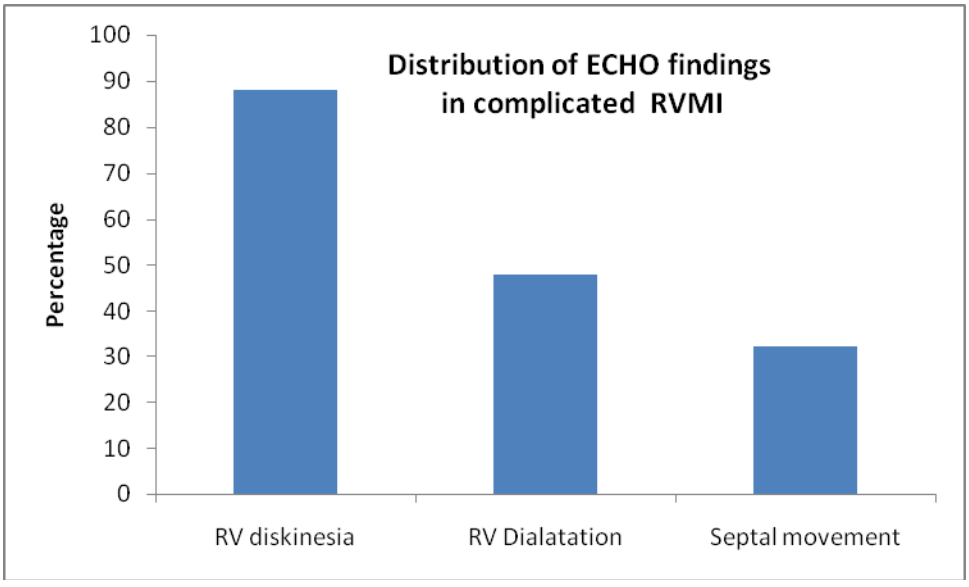
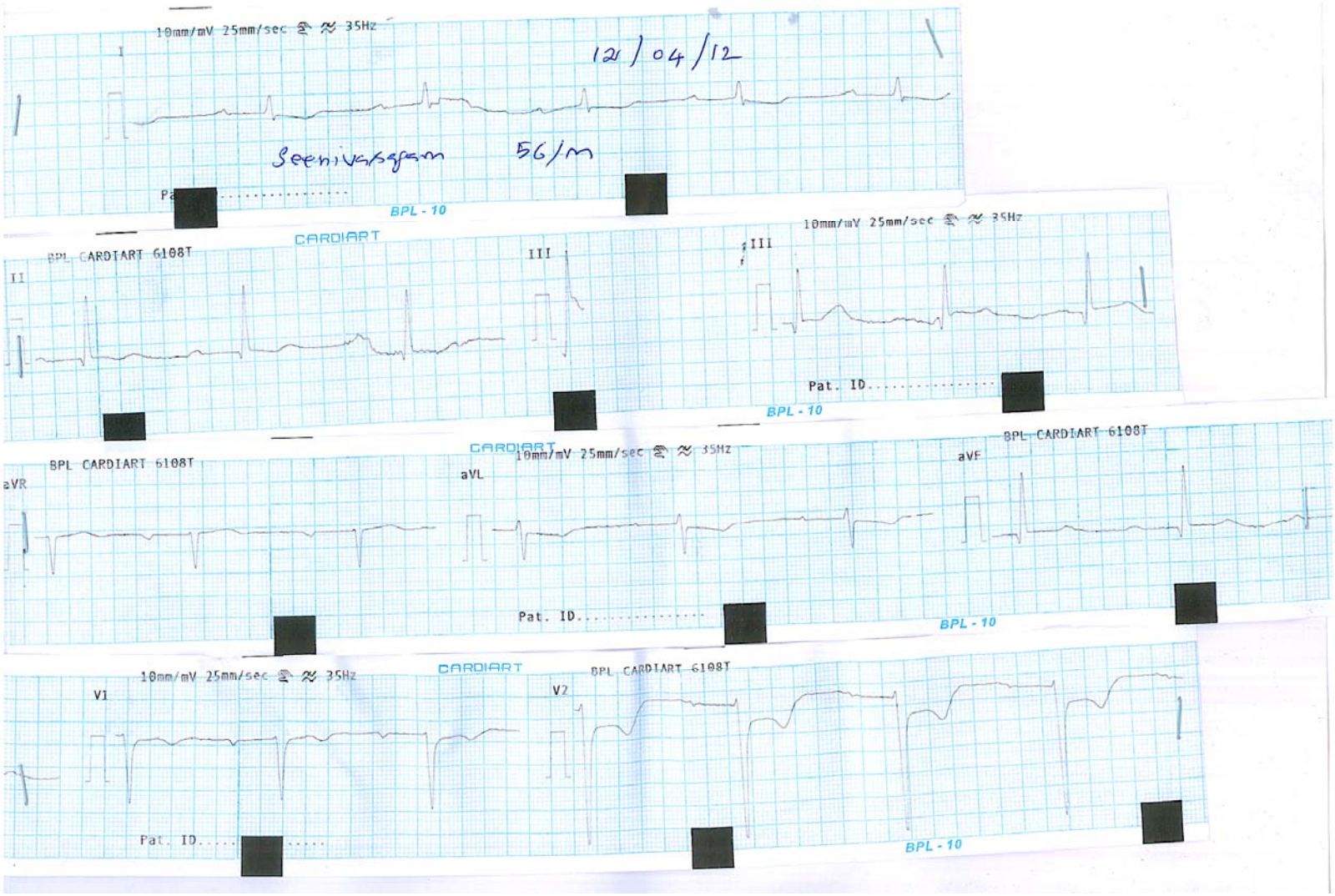


Chart - 6

ECHO FINDINGS AND COMPLICATIONS



ECG 1 – SHOWING IWMI, RVMI, PWMI WITH I^0 AV BLOCK



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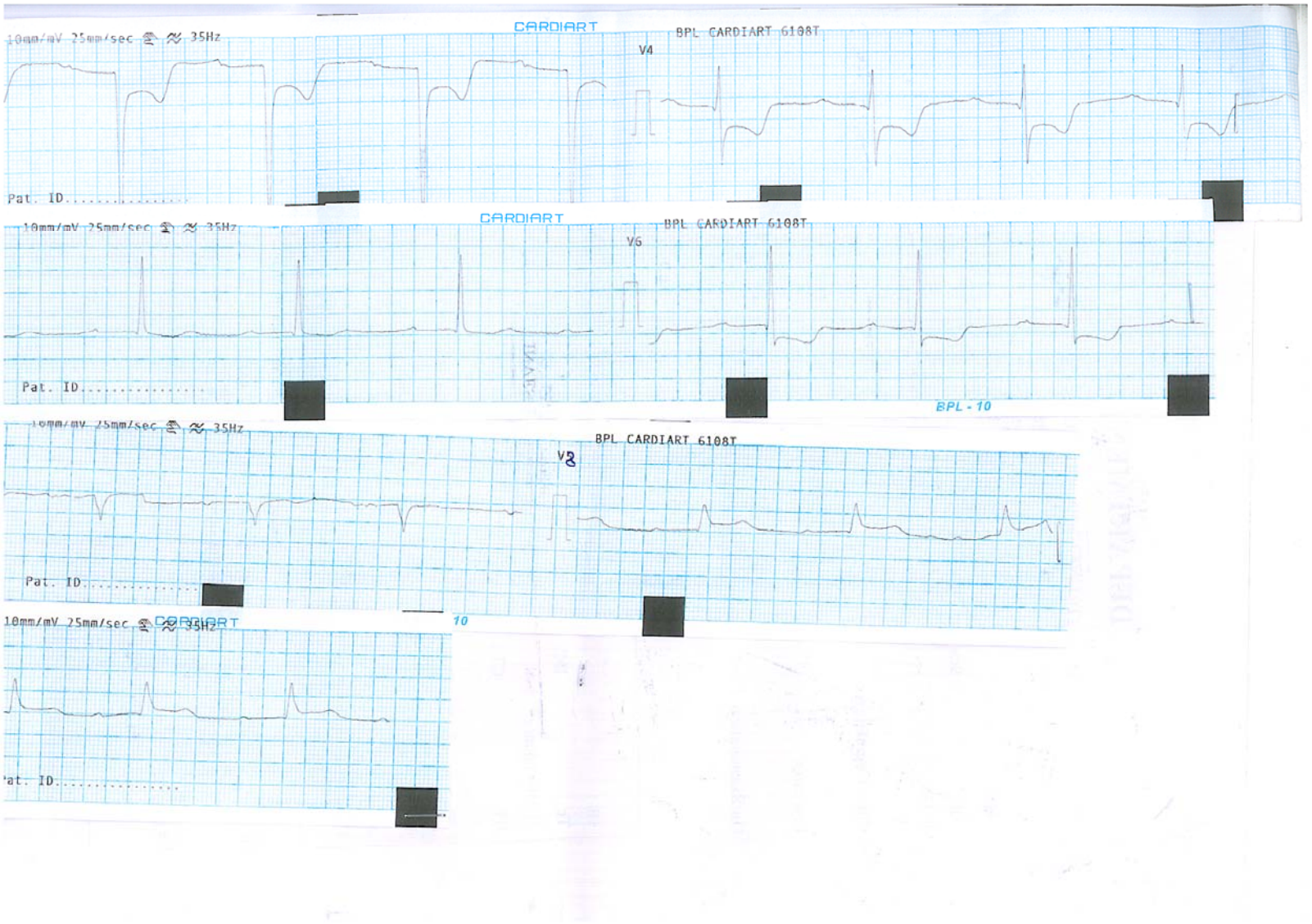
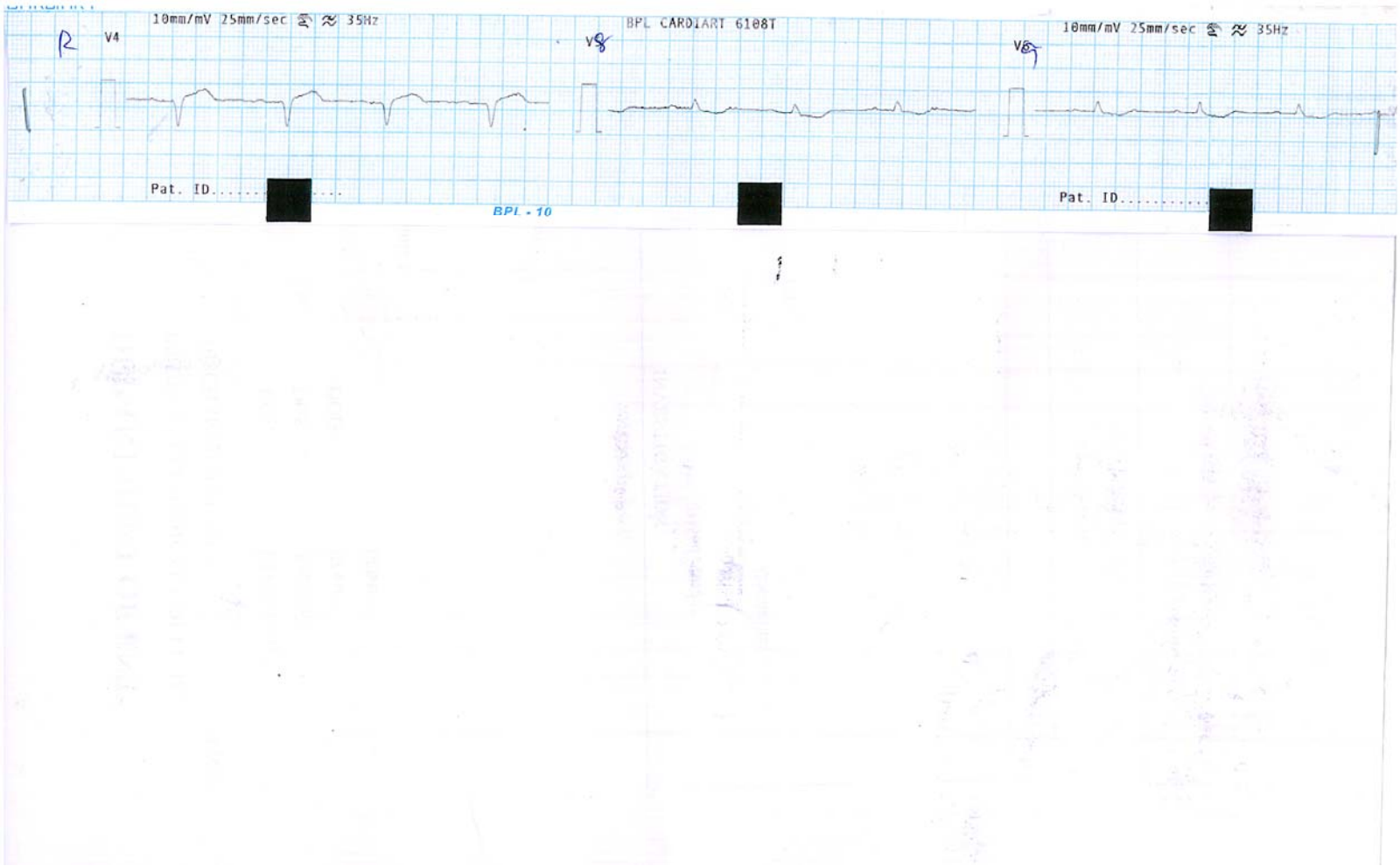

10mm/mV 25mm/sec 35Hz
CARDIART
BPL CARDIART 6108T
V4
Pat. ID.....
10mm/mV 25mm/sec 35Hz
CARDIART
BPL CARDIART 6108T
V6
Pat. ID.....
BPL - 10
10mm/mV 25mm/sec 35Hz
BPL CARDIART 6108T
V5
Pat. ID.....
10
10mm/mV 25mm/sec 35Hz
CARDIART
at. ID.....

ECG 2 – SHOWING IWMI, RVMI, PWMI, AWMI

CONT...


10mm/mV 25mm/sec 35Hz
V4
BPL CARDIART 6108T
10mm/mV 25mm/sec 35Hz
Pat. ID.....
Pat. ID.....
BPL - 10

MASTER CHART

Sl. No.	Age	Sex	Rural / Urban	Occupation	Obesity	DM	HTN	Family h/o CAD	smoking	Symptoms Typical / Atypical	Pain Duration(hrs)	Bradycardia	Hypotension	Elevated JVP	Prominent A wave/ X descent	Shock	Type of MI	ST elevation in V4R	1^0 AV Block	2^0 AV Block	3^0 AV Block (CHB)	RBBB	AF	VT/VF	Death
1	49	M	R	MW			+		+	T	5	+	+	+	+	+	1	>2	+			+		+	
2	48	M	R	MW					+	T	7	+	+	+			1	<2							
3	47	M	U	SL			+		+	T	5	+	+	+	+	+	2	>2				+			
4	57	F	R	HW		+		+		AT	9		+	+			2	<2	+						
5	51	M	R	MW					+	AT	6	+	+				2	<2							
6	53	M	R	MW					+	AT	7	+	+	+	+	+	1	>2			+			+	+
7	62	M	R	MW					+	AT	6	+		+			2	<2							
8	44	M	U	SL			+		+	T	5	+	+	+		+	2	<2	+			+			
9	46	F	R	SL		+		+		T	10						1	<2							

10	45	M	R	MW						T	6		+	+			2	<2							
11	42	M	R	MW			+		+	T	7	+	+				2	<2	+						
12	61	F	R	HW	+	+		+		AT	11	+	+	+	+	+	1	>2				+	+		
13	36	M	R	MW					+	T	5			+			1	<2		+					
14	58	F	R	HW	+	+		+		AT	9	+	+	+			2	<2							
15	46	M	R	MW					+	T	6	+	+				1	<2	+			+			
16	28	M	R	MW					+	T	7			+			1	<2							
17	70	F	R	HW		+				AT	10	+	+	+			2	<2		+					
18	69	F	R	HW		+				AT	11			+			1	<2	+			+			
19	38	M	R	MW					+	T	5	+	+	+	+	+	1	>2							+
20	54	F	R	HW		+		+		AT	3	+	+	+	+	+	1	>2	+		+				
21	42	M	R	S					+	T	9	+	+				1	<2				+			
22	50	M	R	MW	+	+			+	T	13	+		+			2	<2		+					
23	45	M	R	MW					+	T	10	+	+	+		+	2	<2	+						
24	48	M	R	MW					+	T	9			+			2	<2							

25	47	M	R	SL					+	T	11	+	+	+	+	+	2	>2						+	
26	62	M	R	MW					+	AT	10	+		+			1	<2	+			+			
27	54	F	R	S	+	+				AT	13	+	+				2	<2							+
28	52	M	R	MW						AT	9	+	+	+			1	<2							
29	49	M	R	S	+	+	+			T	11	+	+	+			1	<2		+					
30	46	M	U	P	+	+	+			AT	14	+					1	<2	+			+			
31	56	F	R	HW	+	+				AT	15	+	+	+			3	<2	+						
32	55	F	R	S	+	+				AT	14	+	+				2	<2							
33	47	M	R	S	+	+	+			T	13	+	+	+			1	<2	+						
34	33	M	R	MW		+	+		+	T	15		+				3	<2				+			
35	40	M	R	SL	+					T	9	+	+	+			1	<2	+						
36	62	F	R	HW	+	+				AT	13		+	+	+	+	2	>2			+			+	+
37	44	M	U	P	+	+	+		+	T	14						2	<2	+			+			
38	60	M	R	MW		+				AT	10						3	<2							
39	59	F	R	HW	+	+				AT	15	+	+	+	+	+	2	>2	+			+	+		

40	50	F	U	P	+	+				AT	13	+		+			2	<2						
41	46	M	U	P	+	+	+		+	T	5	+					1	<2	+					
42	48	M	R	MW		+			+	T	1	+	+	+	+	+	2	>2				+		
43	32	M	R	MW		+		+	+	T	2	+		+			1	<2	+					
44	53	F	U	SL						T	2			+			1	<2		+		+		
45	57	F	U	S						AT	20	+	+	+			1	<2						
46	60	F	R	HW	+	+				AT	18	+	+	+	+	+	2	>2						
47	64	F	R	HW		+				AT	5			+			2	<2				+		
48	30	M	R	MW			+	+	+	T	3	+	+	+			1	<2	+					
49	69	F	U	HW		+				AT	7	+	+	+	+	+	2	>2						
50	52	M	R	MW		+			+	T	18	+		+			1	<2		+				
51	49	M	R	MW		+			+	T	1		+	+			2	<2	+			+		
52	58	F	U	MW						AT	6						2	<2	+					
53	52	M	R	MW		+			+	T	2	+	+	+			1	<2						
54	60	F	U	MW	+					AT	13	+	+	+			2	<2	+			+		

55	40	M	R	S		+			+	T	3			+			1	<2							
56	49	F	U	P						T	5	+	+	+	+	+	2	>2	+		+				
57	68	F	U	HW	+	+				AT	19	+	+	+			2	<2	+			+			
58	45	M	R	MW		+			+	T	18	+	+	+	+	+	1	>2							+
59	30	M	U	SL	+	+	+	+	+	T	1	+	+				1	<2	+	+		+			
60	64	F	U	MW	+					AT	17	+	+	+	+	+	2	>2				+			

Key to master chart

M-male. P-professionales , HTN –hypertension AF-atrial fibrillation

F-female, S-sedentary life, T- typical symptoms, VT/VF-ventricular

R-rural , SL- skilled lborer AT-atypical symptoms, tachycardia/ventricular fibrillation.

U- urban, HW-house wives, CAD-coronary artery disease

MW-manual worker, DM- diabetes mellitus RBBB- right bundle branch block